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ABSTRACTS FROM THE LITERATURE RELATING TO CONDITIONS OF THE TRACHEA AND BRONCHI.

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This review of the literature is from late 1940 to June, 1941, and was taken from American and English journals. There have been many papers written in great detail on laryngotracheobronchitis, and the majority of them have been abstracted. The remainder of the abstracts deal with Anatomy, Pathology, Different Operative Methods and the Treatment of Conditions and Diseases Pertaining to the Trachea and Bronchi. While not dealing with conditions of the trachea, etc., it was felt that the opportunity should not be missed to comment on "a short resumé of conditions as they existed for the past 50 years, as given by Dean[1] in the Jubilee Volume of the *Annals*. Special stress and emphasis is placed on the importance of the work accomplished by the American Board of Otolaryngology. Also, the work of the teaching section of the American Academy of Ophthalmology and Otolaryngology and the influence this organization has had on otolaryngology is discussed. Because of improved teaching methods, Dean feels that there is no reason why one should go abroad for study to become a competent otolaryngologist."

It has always been felt that with the vast amount of clinical material available in all the larger and even smaller medical centres, the teaching of otolaryngology may be very well conducted within our own country. With the higher standards of medical education which have been formulated within the specialty, and the guidance of the American Board of Otolaryngology, better otolaryngologists will be developed.

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There are no good reasons why the United States cannot assume the rôle of teacher for any of the specialties with the equipment available. Full-time teachers, assisted by able clinicians, will undoubtedly obtain in the future in all our teaching institutions.

Proetz[2] gives a short review of the history of the *Annals*.

The anatomy of the bronchi in relation to technique of bronchoscopy is discussed by Barsby.[3] Postural drainage and methods of maintenance are given in detail.

In the report by Norris, Kochenderfer and Tyson,[4] 22 infants were studied (age from four lunar months to time of birth). They conclude that at birth the epithelial lining of the alveoli (air sacs) contain no visible epithelium. Before the gestation age of five to six lunar months, the obvious epithelial lining of the terminal air sac and the position of the mesenchymal capillaries at a distance from the potential air spaces are a great impediment to respiration.

This modification by Holinger[5] of the distally lighted bronchoscope is an adaptation of the Negus tip, a detachable lamp from the light carrier and the handle of such size to give the instrument better balance — all applied to the original Jackson infant bronchoscope.

This modification by Jesberg[6] of the Tucker 3 mm. bronchoscope, which, in turn, is a modification of the Jackson 4 mm. bronchoscope, has been accomplished by the use of a very small bronchoscopic light bulb. The outside diameter of the 4 mm. size is 6.5 x 5.5, and for the 3 mm. size, 5 x 4.5 mm. These measurements refer to the beak of the instrument. They are very useful instruments for use in infants and are a distinct improvement in the more delicate instruments in the armamentarium.

This very ingenious forceps has been devised by Matis[7] to be used through a special dilating tube. The author advocates its use to control hemorrhage, for the fixation of deep-lying tissue, closing a wound and generally for endoscopic operation.

Matis[8] describes a method of producing a permanent opening in the trachea. The method is indicated in inoperable growths of the larynx or chronic inoperable stenosis of the larynx.

This very short, concise review of the use of anesthesia in peroral endoscopy by Jackson and McReynolds[9] must be read to be appreciated. They have epitomized the subject from their own experiences and combined it with that of many others.

Zavod[10] describes a double channel tube for intrabronchial intubation. Each channel has a separate inflatable bag to permit separate respiration of each lung.

These observers, Lemer, Pinner and Zavod,[11] have found by studies on patients with tuberculosis that neither clinical nor Roentgenological findings are reliable indices of the functional capacity of the lungs. The authors felt that vital capacities were entirely misleading and that bronchospirographic studies should be made in all instances before any irreversible collapse procedure was carried out.

Jackson and Judd[12] studied 137 patients with lung abscess and they felt that the rôle of bronchoscopy should be considered from a threefold standpoint, *viz.: 1.* as a prophylactic measure in the prevention of abscesses because of aspiration of material from the bronchus; *2.* as a differential diagnostic measure; and *3.* as a therapeutic measure in the early stage of abscess to facilitate drainage, etc.

Nail[13] advises that endoscopic operations in children under 12 years be conducted under avertin with amylene hydrate, used alone or with larocaine hydrochloride. For routine bronschoscopy in adults, he makes a local application in the pyriform sinuses and instillates 1 cc. of a 2.5 per cent solution of larocaine hydrochloride and, if necessary, further instillation of the same solution through the bronchoscope.

The old story, as noted by Harner,[14] of the mother insisting that the symptoms shown by the child — paroxysmal coughing, fever and cyanosis — following eating a particular substance, and the family physician probably hoping against hope that the child would cough up the foreign body, if such was the case, is here seen. Because of the somewhat isolated ranch home, the child was not X-rayed until approximately one month had elapsed since the onset of the symptoms. The foreign body was easily removed but, to add to the bronchoscopist's further worries, the temperature became higher

following the removal, and a morbillous eruption appeared. Eventually, after measles had disappeared, the baby, age 22 months, was cured.

Paterson's[15] report is that of a metal end of an eversharp pencil originally in the left main bronchus and a sequence of symptoms following instrumentation. Removal was done at the second attempt by the use of a hook.

The report of McHugh[16] of this case history and the subsequent discussion deserves careful study. It definitely amplifies the contention of the dictum that an X-ray study of the chest is indicated in anyone, regardless of history, where an increased respiratory rate cannot be accounted for. The surgery done on this little patient and the immediate subsequent after-treatment were of the highest order, regardless of complications.

Russ and Strong[17] are of the opinion that intratracheal catheterization is most valuable in asphyxia of the newborn. By alternating inspiration and expiration and aspiration through the tube, great help is given. All other methods are considered and discussed. Intravenous or subcutaneous use of alpha lobeline 1/20 gr., coramine 2 m. and caffeine soda benzoate are considered. Twelve hundred patients were studied and of these, in 196 it was necessary to do resuscitation.

This most unusual case report is given in detail by Schall and Johnson[18] and includes the therapeutic measures instituted and the fatal outcome because of erosion and perforation of the end of the tracheotomy tube into the aorta.

Bozer[19] directs attention to bronchial collapse but, because of no available postmortem observations, discusses the mechanism of this condition from a speculative standpoint. He considers the possibility of an exceedingly strong spasm or a weak bronchial wall inducing collapse and subsequent atelectasis. Case histories of three patients are given.

Ashland and Einstein[20] report a case with the triad of symptoms as first described by Kartagener. They are in accord with Sauerbruch that 80 per cent of cases afflicted with bronchiectasis occurring in childhood are congenital. Their case report was that of a child age 12 years. The author's contention is that the upper respiratory infection

was not primary, although it played a part in the subsequent underlying bronchial disease.

St. Engel and Newns[21] discuss the probability that proliferative mural bronchiolitis is a clinical entity and not previously described. In the condition described, there is an infiltration of bronchiolar wall itself. The authors maintain that the condition begins as a bronchiolar one and not in the alveoli.

This case report by Imperatori[22] deals with an infant, age 7 months, who developed a swelling in the right side of the neck that increased in size so rapidly that the child could breathe only with the head and neck in an extreme position of extension. Diagnosis was made by puncture. A bloody serous fluid that did not coagulate was obtained. Aspiration was further applied and 75 cc. of fluid was removed. The improvement was immediate. The next day 10 cc. of fluid was removed and 3 cc. of a solution of diathame hydrochlor 0.75 per cent, quinine hydrochlor 5.5 per cent, and urethane 3 per cent was injected. A cure resulted.

The basis of this report by Tewksbury and Fenton[23] is the analysis of the results of the treatment of 62 patients with iodized oil, who had either chronic bronchitis or bronchiectasis. Eighty-three per cent of the patients received either complete or partial relief. The author's conclusions are that small doses of iodized oil in the bronchi is a safe procedure in chronic bronchitis. Contraindications given to this treatment are active pulmonary tuberculosis, acute infection or myocardial weakness with pulmonary edema.

The conclusions arrived at by Haggard and Greenberg[24] in a study of the reaction of the mucous membranes of the respiratory tract to mentholated cigarettes are that menthol added to tobacco in the amount now present in cigarettes on the American market exerts no toxic or irritant action.

Myerson[25] reports the results of observations in 29 bronchoscopies among tuberculous children. The ulcerogranulomatous type of lesion frequently seen in adults is not so common in children. Instead, the most common gross lesion is that caused by pressure upon the bronchial wall by enlarged mediastinal lymph nodes. Perforation of these lymph nodes into the bronchus is uncommon.

Wilson[26] endeavored to review present day diagnostic measures and methods of treatment of tuberculosis of the larynx and adjacent areas without attempting to invade the field of the specialist in pulmonary diseases or that of the thoracic surgeon. Most of the observations have been from personal experience over a prolonged period, and he believes that they warrant the statement that worthy progress is being made in the attack on laryngeal, oral, aural and tracheobronchial tuberculosis.

Smith[27] is of the opinion that many pathogenic fungi produce diseases in man which have a higher mortality than tuberculosis. In the treatment of some of these conditions, namely, actinomycosis, iodides were found to be of value. Gentian violet in pulmonary infection by monilia and the use of colloidal copper or antimony and potassium tartrate in coccidioidal granuloma is advised. Sulfanilamide was of value in the treatment of actinomycosis.

Vadala[28] has given a very complete article on mycotic infection of the bronchopulmonary tract. Diagnosis is established by bronchoscopy, aspiration of exudate and further study by cultural and microscopic methods. Many patients are misdiagnosed and commonly considered as tuberculous. Positive diagnosis by clinical observation, repeated negative sputum for tubercle bacilli, bronchoscopic aspiration and the proper treatment other than that for tuberculosis is important in mycotic disease, since in the mycoses the iodides offer the most helpful method of cure, which in tuberculosis should not be used because of its detrimental effect. Proper X-ray interpretation aids greatly in diagnosis. Treatment, in mycotic infections, depends on the organism isolated and is outlined in detail in the different subdivisions of this article. A bronchoscopic department should be included in the clinical setup of all hospitals treating diseases of the bronchopulmonary system.

Connell and Trowbride[29] enumerate and discuss the pitfalls in arriving at a diagnosis of laryngotracheobronchitis and stress the early occurrence of subglottic stenosis. Their recommendation for immediate relief of high obstructive dyspnea is by tracheotomy, sulphotherapy and good supportive care.

Acute laryngotracheobronchitis is a clinical entity, for in infants and young children it is characterized by obstructive dyspnea and requires in most instances instrumental intervention. Kayser[30] is of the opinion that intubation should be used in mild instances and where the procedure can be done by a skilled operator. In other more severe cases where bronchoscopic examination and suction for the removal of plugs or of membrane is necessary, tracheotomy is the more appropriate measure.

This article by Litchfield[31] impresses one with the necessity of early diagnosis and early treatment with warm vapor charged with essential oils. A very careful evaluation is given to treatment when necessary by suction, intubation and tracheotomy.

Cummings[32] reports an acute laryngotracheobronchitis is a relatively uncommon disease, appearing most frequently in infants and children. It tends to spread by continuity of tissue from the larynx and trachea to the smaller airways. The secretions are viscid and, as the disease progresses, there is a tendency to the occlusion of the smaller airways by secretion, crusts and edema. The hemolytic streptococcus is the organism most frequently found, but any of the common bacteria except the diphtheria bacillus may be recovered in pure or mixed culture. Treatment demands bed rest, highly humidified air, adequate fluid intake and, at times, sulfanilamide, tracheotomy or intubation; instillation into the tracheotomy tube to prevent drying of secretions of normal saline, or, if the staphylococcus is present, bacteriophage, and bronchoscopic removal of secretion and crusts. There are here reported the case histories of 14 patients on whom tracheotomy was performed because of the severity of the infection. The mortality was 50 per cent.

Orton, Smith, Bell and Ford[33] give an analysis of 62 cases of acute laryngotracheobronchitis, with reports of autopsies in eight cases. Acute laryngotracheobronchitis in young children is an important problem both for the general practitioner and for the pediatrician. Proper handling of this disease requires early recognition. When signs of obstructive dypsnea appear, valuable time should not be lost in treating the condition with diphtheria antitoxin. When acute laryn-

gotracheobronchitis is suspected, the patient should be transferred immediately to a hospital where direct laryngoscopy and bronchoscopy can be carried out immediately and where more active treatment, such as intubation and tracheotomy, may be done at a moment's notice, with an especially trained personnel always immediately available. If time permits, Roentgen study should be made prior to operation; otherwise, it should be done immediately after tracheotomy. Intubation should be done only for immediate relief; this should be followed by tracheotomy. Patients should be placed in a room in which the air is supersaturated with moisture and maintained at a temperature of 70° F. at all times. This temperature and high humidity provide a more comfortable environment for the patient than the old-fashioned hot croup tent. By the use of a mechanical humidifier, the humidity in a tent can be raised to 95 per cent when the room temperature is 70° F. The temperature and humidity of an oxygen tent have more effect on the comfort of the average patient than does the concentration of oxygen. If the temperature of the air is not above 70° F., the loss of water from the body can be decreased by increasing the humidity. Tracheotomy is the operation par excellence.

Wilson[34] and his associates at Willard Parker Hospital, New York, states: "We consider as indicated for tracheotomy those cases in which obstruction is not relieved by intubation, and cases in which we cannot extubate after one week. This statement needs some explanation. In diphtheritic croup, the patients recover or die within a few days, and an intubation tube is inserted for only a short period. In laryngotracheobronchitis, the patients do not recover so rapidly, although they frequently die rapidly; therefore, they require help in breathing for one or two weeks or more. The pressure of an intubation tube upon a succulent, tender, inflamed mucous membrane produces ulceration, and when these are deep the resulting scar formation causes chronic laryngeal obstruction. In laryngotracheobronchitis, cyanosis is not common because the obstruction does not occur suddenly or completely but is slow in development and incomplete. In these cases of slowly progressive obstruction of the larynx there is a high pitched inspiratory rasp accompanied by retraction of the supraclavicular region, the spaces between the ribs and the sub-

sternal region. The face is pale and not cyanosed; the lips are pale; and as the dyspnea becomes more severe the pallor of the face increases, and a white line appears on the upper and lower lips, completely surrounding the mouth. This is a terminal sign indicating impending death through exhaustion and lack of oxygen and is an imperative indication for immediate tracheotomy. Sudden death occurs following tracheotomy, both in adults and in children, by the sudden dilution of the residual carbon dioxide in the lung by the inrush of air. The remedy is the administration of carbon dioxide and oxygen, and this should be on hand and ready for use alongside of the operating table. It is best to administer carbon dioxide and oxygen to the patient as a routine immediately following a tracheotomy because if one waits for symptoms, the patient has already ceased breathing from lack of carbon dioxide, and it is then difficult, if not impossible, to get sufficient carbon dioxide to the respiratory centre in time to revive the patient.

Emphysema and Pneumothorax: This is common, occurring in about 75 per cent of the cases. When pneumothorax occurs on both sides, it is exceedingly dangerous. It may occur spontaneously, with or without a tracheotomy.

Halpert[35] emphasizes that in his experience, males between the ages of 41 and 60 years comprise the majority of patients with carcinoma of the lung. The site of the origin is in a bronchus or one of its branches, in either lung. In those instances where the site of the lesion is easily seen through the bronchoscope because of the proximity of the bifurcation of the trachea, the possibility of operation is lessened, whereas those lesions in the smaller branch bronchi and inaccessible to bronchoscopy were more amenable to surgical treatment.

Holinger and Radner[36] conclude that bronchoscopy in bronchial carcinoma are fourfold: *a.* To study the character of the lesion; *b.* to note its location, etc.; *c.* to secure tissue for biopsy; *d.* to aid in determining the operability of the tumor.

Singer[37] concludes that the clinical picture of carcinoma of the lung is still so varied that no definite pattern has been discovered to fit the many types of pathologic conditions present. Because of the various methods of diagnosis, few cases are now overlooked; also, because the value of diagnosis is

appreciated in order that early surgery can be done. He has described a thoracoscope of his own devising.

Ormerod[38] reviews a series of cases, discusses and considers that there is much for further investigation in differentiating between carcinoma and so-called adenomata. Diagnoses in the cases that he has reviewed were all established (histologically). The results with the use of radon seeds are tabulated. Twenty-one patients survived for more than one year after treatment but with only one alive after the second year.

Samson,[39] in discussing Jackson and Judd's[12] paper before the 1940 meeting of the American Society of Thoracic Surgery, stated that, next to cancer, lung abscess was the most fatal common disease of the chest, and advocated bronchoscopic examination in all patients excepting those who had a brain abscess or the complication was hemorrhage.

Poppe[40] gives an epitome, in lung abscess, bronchiectasis and lung tumor, of the method of arriving at a diagnosis, etiology and pathology and symptomatology. Also, the indications for the surgical treatment of lung abscess. Lobectomy in bronchiectasis and pneumonectomy for lung tumor.

Moore[41] evaluates this method of radiology (laminography), stating that in his opinion early diagnosis of malignant tumor will be accomplished through this method.

BIBLIOGRAPHY.

1. Dean, L. W.: Fifty Years of Otolaryngology in America. *Ann. Otol. and Rhinol.*, 50:1, 1941.

2. Proetz, A.: The Annals, 1892-1941. *Ann. Otol. and Rhinol.*, 50:8, 1941.

3. Barsby, B. E., and Bonham-Carter, R. E.: The Diagnosis and Conservative Treatment of Bronchiectasis in Children. *Arch. Dis. Childhood*, 16:95, 1941.

4. Norris, R. F.; Kochenderfer, T. T., and Tyson, R. M.: Development of the Fetal Lung. *Am. Jour. Dis. Child.*, 61:933, 1941.

5. Holinger, P.: New Infant Bronchoscope and Esophagoscope. *Arch. Otolaryngol.*, 33:431, 1941.

6. Jesberg, S.: Baby Bronchoscopes. *Arch. Otolaryngol.*, 33:88, 1941.

7. Matis, E. I.: Method of Suturing Through an Endoscopic Tube. *Arch. Otolaryngol.*, 33:316, 1941.

8. Matis, E. I.: Plastic Tracheostomy. The Laryngoscope, 51:565, 1941.

9. Jackson, C. L., and McReynolds, G. S.: Anesthesia for Peroral Endoscopy. *Ann. Otol. and Rhinol.*, 41:1048, 1940.

10. Zavod, William A.: Bronchospirography. *Surg.*, 8:889, 1940.

11. Lemer, George; Pinner, Max, and Zaved, William A.: Bronchospirography; Application to Collapse Therapy. *Surg.*, 8:889, 1940.

12. Jackson, C. L., and Judd, A. R.: The Role of Bronchoscopy in the Treatment of Pulmonary Abscess. *Surg.*, 8:893, 1940.

13. Nail, J. B.: Anesthesia in Peroral Endoscopy. *Tex. State Jour. Med.*, 36:430, 1941.

14. Harner, C. E.: Vegetable Foreign Body in the Lung for a Month. Successful Removal. Recovery Complicated by Measles. *Arch. Otolaryngol.*, 32:951, 1940.

15. Paterson, N.: An Unusual Method of Dealing with a Foreign Body in the Lung. *Jour. Otol. and Laryngol.*, 56:65, 1941.

16. McHugh, H. E.: Foreign Body in the Lung. *Arch. Otolaryngol.*, 33:1100, 1941.

17. Russ, J. D., and Strong, R. A.: Resuscitation of the Asphyxiated Newborn Infant. *Am. Jour. Dis. Child.*, 61:1, 1941.

18. Schall, L. A., and Johnson, L. G.: Dyspnea Due to Congenital Anomaly of the Aorta. *Ann. Otol. and Rhinol.*, 41:1055, 1940.

19. Bozer, H. E.: Bronchial Collapse Without Clinical Signs of External Pressure. Bronchoscopic Observations. *Ann. Otol. and Rhinol.*, 49:1091, 1940.

20. Adland, S. A., and Einstein, R. A. J.: Kartagener's Triad (Situs Inversus Viscerum, Bronchiectasis and Paranasal Sinusitis). *Am. Jour. Dis. Child.*, 61:1034, 1941.

21. St. Engel and Newns, G. H.: Proliferative Mural Bronchiolitis. *Arch. Dis. Childhood*, 15:219, 1940.

22. Imperatori, C. J.: Hygroma Cysticum Colli. *Trans. Am. Laryngol. Assn.*, 62:51, 1940.

23. Tewksbury, W. D., and Fenton, E. R.: The Therapeutic Use of Iodized Oil in Chronic Bronchitis and Bronchiectasis. *Diseases of Chest*, 7:20, 1941.

24. Haggard, H. W., and Greenberg, L. A.: Concentration of Menthol in the Smoke from Mentholated Cigarettes, etc. *Arch. Otolaryngol.*, 33:711, 1941.

25. Myerson, M. C.: Bronchoscopy in Tuberculous Children. *Ann. Otol. and Rhinol.*, 49:1064, 1940.

26. Wilson, G. E.: Diagnosis and Treatment of Tuberculosis of the Larynx and Contiguous Areas. *Arch. Otolaryngol.*, 33:145, 1941.

27. Smith, T. David: Treatment of Fungus Infections of the Lung. *Surg.*, 8:888, 1940.

28. Vadala, A. J.: Mycotic Infection of the Bronchopulmonary Tract. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 46:275, 1940.

29. Connell, E. S., and Trowbridge, B. C.: Acute Streptococci Laryngotracheobronchitis. *Arch. Otolaryngol.*, 33:717, 1941.

30. Kayser, B.: Acute Laryngotracheobronchitis. Report of a Case. Review of the Literature. *Arch. Otolaryngol.*, 33:795, 1941.

31. Litchfield, H. R.: Treatment of Acute Obstructive Infections of the Larynx, Trachea and Bronchi. *Arch. Ped.*, 57:717, 1940.

32. Cummings, G. O.: Acute Laryngotracheobronchitis with Reports of 14 Cases Which Came to Tracheotomy. The Laryngoscope, 51:382, 1941.

33. Orton, H. B., and others: Acute Laryngotracheobronchitis. *Arch. Otolaryngol.*, 33:926, 1941.

34. Wilson, A. S.: Acute Contagious Diseases — Upper Respiratory Complications. The Laryngoscope, 50:48, 1940.

35. Halpert, Bela: Morphologic Aspects of Carcinoma of the Lung. *Surg.*, 8:903, 1940.

36. Holinger, P., and Radner, D. B.: Bronchoscopic Diagnosis of Bronchial Carcinoma. *Surg.*, 8:939, 1940.

37. Singer, J. J.: Primary Bronchiogenic Carcinoma. *Surg.*, 8:910, 1940.

38. Ormerod, F. C.: Some Notes on the Treatment of Carcinoma of the Bronchus. *Jour. Laryngol. and Otol.*, 56:1, 1941.

39. Samson, P. C.: Discussion — Jackson and Judd's paper (see Ref. 12). *Surg.*, 8:893, 1940.

40. Poppe, J. K.: Lung Abscess, Bronchiectasis and Lung Tumor. *Am. Jour. Nursing*, 1:11, 1941.

41. Moore, Sherwood: Body Section. Radiography in Malignancy of the Lower Respiratory Tract. *Surg.*, 8:924, 1940.

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A PRESENT CONCEPTION OF THE GRADENIGO SYNDROME.*

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In 1904 and 1907, Gradenigo wrote his classic papers on the syndrome named for him. This consisted of the triad, pain in the head, especially in the temporoparietal region, purulent otitis and VIth nerve paralysis, all on the same side of the head. These papers stimulated an intensive study of the petrous bone and resulted in greater knowledge of the latter. its semiology, histology, pathology and especially the surgical technique for its drainage when necrosis occurred in different parts of the petron.

The production of this syndrome generally starts as an acute infective otitis. Since the tympanum has a direct connection with the air cells of the mastoid and of the petrous pyramid, an infection of the one involves to a lesser or greater degree the others.

Sometimes it is the mastoid cells which succumb to the infection, and a coalescing osteitis may result, requiring a mastoidectomy.

At other times the main infection is in the petrous cells, and if the bone necroses, drainage may be necessary.

The third condition may involve air cells in both the petron and the mastoid, which is most common in Gradenigo's syndrome.

In a very large percentage of these infections of the tympanum, only the mucosa of the tympanum, the mastoid and petrous air cells are involved; there is little or no destruction of the cell walls; the bacterial toxins may pass from the subepithelial cells through the tissue spaces, through the bone to the periosteum or dura near the air cells, particularly where the bone is thin or deficient. Inflammatory edema results in these areas, which may subside promptly after free drainage is secured by a myringotomy.

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If the edema occurs at the apex of the pyramid near Dorello's[1] canal, the swelling of the dura will so constrict the abducent nerve that paralysis results.

If the infection does not cause much, if any, destruction in the petrous cells, and if the mastoid cells when they necrose are removed, the pain and VIth nerve paralysis promptly disappear.

This is the usual history of what should be called the Gradenigo syndrome.

The condition is that of subacute petrositis or petrosismus, as Pragier[2] suggested in 1933.

The appearance of the pain and diplopia is early, usually within four weeks, the X-rays may show some cloudiness of the petrous air cells but no destruction of the bony cellular walls. The constitutional symptoms are mild in subacute petrositis *per se*.

PAIN.

The pain is at first intermittent and worse at night, later becoming more constant and agonizing.

Gradenigo localized it usually in the temporoparietal region, though at times around the forehead, orbit, teeth and neck.

There are many attempted explanations for its cause.

1. Pressure or irritation of the Gasserian ganglion in the cavum Meckeli, where one layer of the dura separates the ganglion from an apical inflammation. This layer of dura is closely adherent below to the bone of the apex, and above to the ganglion.

Inflammatory edema of the dura beneath the ganglion is supposed to exert sufficient pressure on the three branches of the Vth nerve to cause reflex pains in the various distributions of the trifacial.

2. Vail[3] suggested that the greater superficial petrosal nerve could easily be affected by inflammation at the apex as it is close to the petrous apex, from the hiatus Fallopii along near the crest of the pyramid, passing under the trifacial ganglion and finally reaching the lacerated foramen,

where it joins the great deep petrosal nerve to form the nerve of the pterygoid canal (Vidian nerve); but it is yet to be proven that there are nerve fibres of pain sense in this nerve. Moreover, the irritant is applied to the nerve and not to the nerve sense terminations, and so would need greater stimuli to produce pain.

The greater superficial petrosal nerve enervates the lacrimal gland, so disease of it decreases the lacrimal secretion.

Tremble and Pennfield[4] reported a case of perineural fibroblastoma of the nerve without any pain, but there was decreased secretion of the lacrimal gland on that side.

Seydell[5] reported 41 cases of petrositis, 36 per cent of whom had no characteristic pain, and 5 per cent had no pain.

3. It is generally agreed among physiologists that cephalgia usually results from stretching of the dura, either locally or generally.

Head,[6] in explaining reflex pains, stated that when a painful stimulus is applied to a part of low sensibility in close connection with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility rather than in the part of lower sensibility to which the stimulus was actually applied. Thus, in irritation of the dura the pain is switched to the more sensitive areas supplied by the same nerves, such as the parietotemporal, eye, teeth, etc., regions.

Krause, in 1904, suggested that the pain was due to dural irritation, and this was mentioned by Myerson[7] in July, 1935.

The tentorium is enervated by a recurrent branch of the first or ophthalmic division of the trifacial. The tentorium is attached to the crest of the pyramid, to the posterior and anterior clinoid processes, and could easily be stretched by inflammatory edema near the apex, resulting in a reflex pain near the eye.

The dura of the middle fossa is enervated by branches from the maxillary and mandibular nerves of the trifacial. The stretching of the dura on the anterior surface of the pyramid near the apex could easily cause reflex pains in the temporoparietal region, in the jaws, teeth and face.

The dura of the posterior fossa is supplied by branches from the vagus and hypoglossal. Its stretching could readily produce reflex pain in the occipital and cervical regions.

It would appear far easier to produce afferent impulses in the receptive terminal sensory endings of a nerve of pain sense than in the nerve trunk, surrounded as the latter is with neurilemma, capillaries and perivascular tissue spaces. Only in confined bony or strong ligamentous spaces would it be logical to expect that sufficient pressure would be applied to initiate afferent pain impulses.

Inflammation of the nerve sheaths producing a neuritis might conceivably cause sufficient pressure to cause pain.

In the Gradenigo syndrome the pain disappears too quickly to consider it to come from an infectious neuritis.

There have been several cases reported where the Gasserian fibres were bathed in pus without any pain symptoms. Friesner[8] quotes the cases of Uffenorde, Hilgerman and Brunner.

Some of the nerve fibres here showed degeneration, but there were surely sufficient fibres remaining normal to transmit pain sensations if caused by neuritis.

Another explanation for the pain in and around the eye is that the ophthalmic nerve is closely bound down by the dura and would, therefore, be put on a stretch when the latter swells. It is also stated that the two other branches of the Vth are not bound down tightly.

How, then, explain the temporoparietal, teeth, etc., pain?

ANATOMY.

A brief survey of the anatomy of the petrous apex region follows:

The tentorium divides to enclose the lateral sinus posteriorly; anteriorly, it is attached to the ridge of the petrous pyramid enclosing the superior petrosal sinus, then passes medially to the posterior clinoid process.

The free border of the tentorium passes forward to the anterior clinoid process, crossing the part that goes to the posterior clinoid process.

It also helps to form the dural diaphragm over the sella.

The tentorium is very adherent to the petrous ridge and the clinoid processes and helps to form the upper boundary of Dorello's canal.

The inferior petrosal sinus passes from the cavernous sinus posteriorly to Dorello's canal, where it is usually situated medially and more or less above the VIth nerve. It then enters a groove along the petro-occipital suture, passing outward and backward to the jugular foramen, finally ending in the internal jugular vein.

The VIth nerve has the longest free course in the cranium of all the cranial nerves.

Its superficial origin is at the lower edge of the pons, at about the junction of the olive and the pyramid.

It is only 6 mm. separated from the VIth nerve of the opposite side at this point.

The nerve then passes upward and slightly outward for a distance of about 15 mm. to Dorello's canal, crossing usually over the inferior petrosal sinus to the lateral angle of Dorello's canal, then through the canal; then, bending slightly downward, pierces the cavernous sinus, passing forward just lateral to the internal carotid artery.

On leaving the cavernous sinus it passes forward, entering the superior orbital fissure just above and lateral to the ophthalmic vein and below and internal to the inferior division of the IIIrd nerve, the nasociliary nerve and the superior division of the IIIrd. finally reaching the external rectus muscle.

The distance between the two VIth nerves at Dorello's canals is about 20 to 25 mm.

The position of the nerve in Dorello's canals varies a great deal, and the diameters of the canal openings are not uniform. These vary not only in different individuals but also in the same person on the two sides.

Sometimes there is a bony partition between the inferior petrosal sinus and the nerve, thus further limiting the dilatability of the opening for the VIth nerve.

All these variations explain why the VIth nerve palsy so seldom occurs, even in the probable presence of an inflammatory edema.

The trifacial nerve pierces the dura about 10 mm. above the VIth nerve and a little more lateral and close to the roof of the tentorium, passes over the crest of the petrosa near the apex tip, is surrounded by dura and arachnoid which forms a sack, called the cavum Meckeli, which lodges the semilunar ganglion.

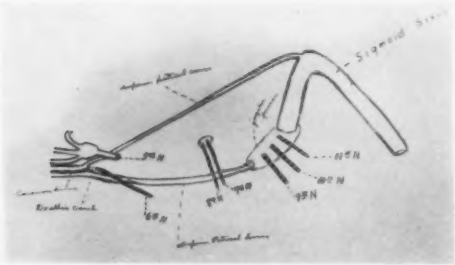

Fig. 1. Showing relations of VIth nerve at Dorello's canal.

The dura below and above the ganglion is closely adherent. The bone under the ganglion is hollowed out into a distinct depression for the ganglion, is usually very thin, and forms the roof for the internal carotid and sometimes for a large pneumatic cell.

The ganglion lies over its motor root and over the greater and lesser superficial petrosal nerves to which it is closely attached.

The motor root becomes incorporated wholly in the mandibular or third division of the Vth nerve.

The ophthalmic nerve is said to be more bound down by the dura and, therefore, more subject to pressure or stretching (Roberts[9]).

Dorello's canal is situated at the synchondrosis of the petrous apex and the basisphenoid, is triangular or crescent-shaped, 8 mm. wide and 4 mm. deep (Profant[10]), with great variations.

The superior petrosal sinus leaves the crest of the petron a few millimetres before reaching the tip of the apex, passes forward and inward, and enters the cavernous sinus anterior to Dorello's canal and to the entrance of the inferior petrosal sinus.

The crest of the pyramid is continued inward to the tip, where it sometimes becomes a projecting process, called the sphenoidal spine.

From the spine a fibrous ligament, reinforced by tentorial dura, extends inward and is attached to the posterior clinoid process, forming the roof of Dorello's canal, and is called the petrosphenoidal or Gruber's ligament.

The lateral wall is formed by dura and the tip of the apex.

The internal wall is the edge of the dorsum sellae of the sphenoid.

The floor is formed by the petrosphenoidal synchondrosis and part of the dorsum sellae of the sphenoid.

The canal is entirely lined with dura and extends forward and slightly upward for a distance of around 4 mm. The VIth nerve is held snugly in a depression just mesial to the tip of the petrous apex.

The sphenoid sinus is sometimes very close, 2 mm., to Dorello's canal, varying according to the size of the sinus. When the sinus is small it may be 22 mm. from the canal.

This explains the occasional VIth nerve paralysis with purulent sphenoidal sinusitis according to Houser.[11]

PNEUMATIC CELLS.

It is the exception to find pneumatic cells at the apex. At birth there is practically no apex beyond the labyrinth.

A well pneumatized mastoid in an adult is usually accompanied by a well pneumatized petrous bone.

The adult petrous is reached in the fourth or fifth year according to Profant.[10]

Pneumatization of apex cells is 10 to 15 per cent, but the percentage is much higher in the cells just medial to the superior semicircular canal.

The cells of the petrous are usually mixed pneumatic and diploic in the adult.

Mastoid pneumatization is usually complete at age 5 years according to Gordon Wilson.[12]

The apex usually pneumatizes later than the mastoid (Friesner[13]) and may continue to penumatize in the pyramid after it has stopped in the mastoid.

Gradenigo found air cells in tip of most cases showing his syndrome.

According to Guild,[14] air cells originate from four primary regions: *1.* Osseous portion of Eustachian tube; *2.* hypotympanum; *3.* epitympanum; and *4.* mastoid antrum.

Marvin Jones[15] found no air cells in 16 specimens in antelabyrinthine region.

Air cells are lined with pavement epithelium and a richly vascular areolar tissue.

Diploic cells are in various proportions in the petrous bone.

In the young they are predominantly red, hematopoietic; in the adult, fatty or yellow.

The amount of red may be greatly increased by local or general infections, changing from fatty to red bone marrow.

The marrow spaces communicate freely according to Gordon Wilson[16] and are more resistant to infection than air cells.

Growing bone has many more blood vessels than the comparatively stationary adult bone.

Diploic cells verge with those of basisphenoid and basioccipital in many adult bones.

The dura is supplied by sensory branches from all three divisions of the Vth nerve.

The nervus tentorii is a small recurrent branch from the ophthalmic trunk near the Gasserian ganglion and, running backward across the IVth nerve, ramifies between the layers of the tentorium.

The ophthalmic nerve, through its lacrimal nerve branch, supplies branches to the lacrimal gland, conjunctiva, and to

the skin of the lateral commissure of the eye. It communicates with the zygomatic branch of the maxillary nerve, which carries secretory fibres to the lacrimal gland from Meckel's ganglion and the greater superficial petrosal nerve.

The frontal nerve from the ophthalmic, enervates through the supraorbital nerve, the skin above the eye.

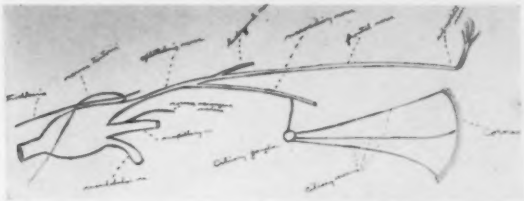

Fig. 2. Showing meningeal branches near Meckel's ganglion and branches to cornea, forehead, etc.

It supplies sensory fibres above the eye and can account for the pain in that region. The nasociliary nerve branch of the ophthalmic, through the ciliary ganglion, enervates the conjunctiva, cornea, iris and iris muscles.

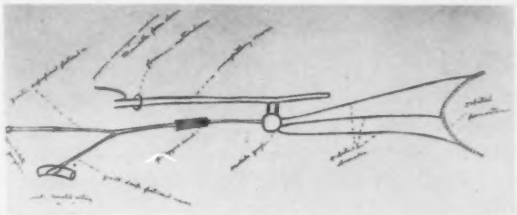

Fig. 3. Showing branches from maxillary nerve through Meckel's ganglion to the orbital periosteum.

The maxillary nerve gives off a recurrent branch near the Gasserian ganglion, the nervus meningeus medius, which passes to the dura and middle meningeal artery, supplying the middle fossa dura.

The maxillary nerve also sends sensory branches to the sphenopalatine ganglion, some of which continue as ascending orbital branches and supply the periosteum of the orbit.

Therefore, pains in and around the eye are enervated by the ophthalmic and maxillary nerves, and can be initiated by stretching of the dura in the middle fossa.

The mandibular nerve gives off, as it issues from the foramen ovale, a recurrent branch (nervus spinosis) which re-enters the skull through the foramen spinosum and supplies the dura in the middle fossa.

The vagus nerve has a recurrent or meningeal branch from the jugular ganglion, which passes upward through the jugular foramen, to be distributed to the dura in the posterior fossa.

Irritation of the dura in the posterior fossa might cause pain in back of auricle and the external acoustic meatus.

The hypoglossal nerve also has a recurrent branch near its origin, which supplies the dura of the posterior fossa at the base of the skull.

This recurrent branch probably derives its fibres from the first and second cervical nerves, and the fact that the great auricular, lesser occipital and greater occipital nerves also arise partly from the second cervical nerve would explain the pain back of the ear and back of head.

PATHOLOGY.

The petrous bone, like other bones in the body, has Harversian and Volkmann canal systems for bringing blood to the cells and removing it through the veins. The blood vessels come from the periosteum, the dura and through the air cells route, in the submucosal lining from the tympanum, mastoid antrum and mastoid pneumatic cells.

The tissue fluids around the blood vessels and the cells are the ultimate intermediaries for bringing fresh products to the cells, and the removal of discarded chemical and biochemical materials.

The infection usually starts in the nasopharynx and Eustachian tubal lining, proceeding thence to the tympanum and the air cells of the mastoid and petron.

The lining membrane of epithelial cells and subepithelial fibrous cells swell and there is a production of toxins, and there may be a purulent exudate in the cell cavities.

In parts there may be destruction of the epithelium. At any rate, the toxins get into the submucosal tissue fluids.

Thence they pass through the bone, either in tissue fluid channels in the canaliculi or in the perivascular or perineural channels to the epidural spaces.

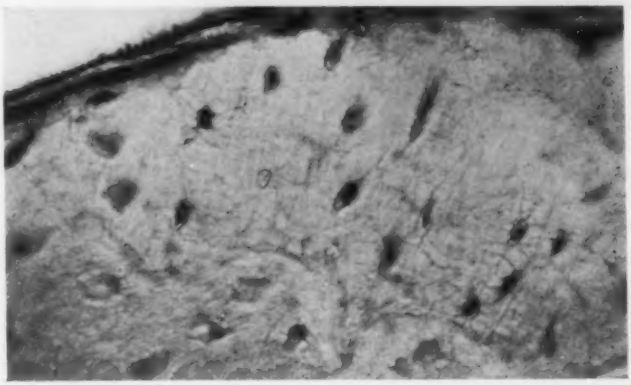

Fig. 4. Showing minute channels and canaliculi in the bone. Obtained through courtesy of Dr. E. P. Fowler, Jr.

The response of the dura to these toxins is inflammatory edema. This is brought about by the histamine and histamine-like substances which cause dilatation and increased permeability of the capillaries, the plugging of the tissue fluid channels, etc., with the result that the dura swells up to several times its normal thickness.

When this dural swelling occurs in confined passages such as Dorello's canals, the pressure may be sufficient to paralyze the VIth nerve by interfering with the circulation of fluids in its perineural envelope.

That it is merely a temporary circulatory phenomenon is proven by the fact that the paralysis often disappears in a day on reduction of the dural swelling.

If the paralysis were due to a neuritis, this could not happen so quickly.

That the inflammatory edema of the dura contains few if any bacteria can be postulated for the same reason; namely, that the process would become purulent otherwise.

The production of abscess over bone is usually preceded by an osteitis or osteomyelitis and direct communication through a dehiscence.

The infection passes by the routes of least resistance; namely, the chains of air cells which were outgrowths from the tympanum, aditus, antrum or mastoid cells such as the peribulbar cells.

In osteitis of the petron, the infective process is slower, so that there may be thrombophlebitis or thromboses in the veins, or even arteritis with thrombosis, the infection proceeding by these paths, as well as the tissue fluid spaces.

Eagleton[17] has shown that these infected thromboses leading through the dura and even into the brain are centres for the production of local abscesses.

VIth nerve paralysis and pain has been reported by Friesner in a case of epidural abscess over the mastoid tegmen. The stretching of the tentorial dura could account for the pain, and the existence of a subacute petrositis, together with inflammatory edema at the apex, could cause the abducent paralysis.

In osteitis of the apex the inflammatory edema is more liable to be localized at the spot where the dehiscence is forming on account of the commonly lengthy destructive process.

Time enough is given for the production of a protective wall of resistance to an extension of the inflammatory edema to Dorello's canal.

This may explain the absence of abducent paralysis in many of these severe cases of bone destruction.

Heterolateral paralysis of the VIth nerve may be explained as follows:

1. Spread of the inflammatory edema to other side of basisphenoid, especially with that Dorello canal being small.

2. Sphenoiditis of the other side.

3. Sinus thrombosis of the other side.

4. Markedly increased intracranial pressure.

5. Undiscovered subacute petrositis of other side.

Of course, other common causes of this paralysis must be eliminated in making the diagnosis, such as syphilitic or tubercular basilar meningitis, tumors, pontine disease, etc.

The VIth nerve has been reported absent on one side.

Perkins[18] stated that in 101 cases of meningitis there were only four with VIth nerve paralysis, or less than 4 per cent.

DIAGNOSIS.

The differential diagnosis between a subacute petrositis with pain, and VIth nerve paralysis; and acute osteitis of the petrous bone while the latter is still intrapetrosal is not easy. In the former, the early appearance of the abducent paralysis compared to the late or absent paralysis in the true case of osteitis; the mere cloudiness of the X-ray of the apex compared to disappearance of cell outline due to halisteresis, especially if there has been a series of pictures taken at two or three angles and the suspected osteitis shows a progression of the destruction.

The rapid improvement, disappearance of pain and lessening of the abducent paralysis after myringotomy or simple mastoidectomy when there is mastoid osteitis clinches the diagnosis, providing there is no recurrence of the Vth and VIth nerve symptoms.

Increased calcium in the pus from the ear may appear in cases of osteitis.

Friesner[19] says that it may contain as much as 37 mg. of calcium per 100 cc.

Of course, this is only of value when the osteitis is confined to the petrous bone.

After the osteitis has become peripetrosal, other symptoms may appear — signs of meningeal irritation, increased cerebrospinal fluid cell count, and changed chemical composition, and possibly increased pressure.

Pain may appear in the occipital or cervical region in the posterior fossa lesions, or symptoms from the IXth, Xth and XIth nerves, such as difficulty in swallowing and hoarseness.

Anterior fossa symptoms such as pain in, around and back of eye, pain in parietotemporal region, teeth, etc. — carotid

plexus symptoms; *e.g.*, Horner's syndrome, such as myosis, ptosis and flushing of same side of face — greater superficial petrosal nerve symptoms, such as decreased lacrimal gland secretion and corneal hyper- or hyposensitivity, in involvement of the ophthalmic nerve through the ciliary ganglion.

Anesthesia of the cornea will occur when the sensory fibres of the ophthalmic nerve that passes through the nasociliary branch to the ciliary gangion are not functioning.

Eye ground symptoms should suggest thrombophlebitis with thrombosis. According to Lillie,[20] this occurs in 10 per cent of lateral sinus thrombosis.

Paralabyrinthine symptoms may occur when the osteitis is near the labyrinth, and facial nerve symptoms when the osteitis is near the internal auditory meatus or the Fallopian canal.

Lateral pharyngeal symptoms may appear when pus breaks through the inferior surface of the apex or through the jugular foramen.

Loss of sensation, thermal and tactile, in the areas of the Vth nerve distribution, may occur in severe involvement of the Gasserian ganglion or its nerves.

TREATMENT.

The treatment of subacute petrositis without osteitis resolves itself into that of the tympanum, and the mastoid if there is any osteitis in the latter.

An efficient myringotomy and, when necessary, a thorough simple mastoidectomy, removing all the air cells in the zygomatic, the semicircular canals and the peribulbar regions, will usually result in the quick disappearance of the pain and improvement of the abducent nerve paralysis.

Chemotherapy should be given intensively as long as there is no osteitis of the petrous bone.

CONCLUSIONS.

1. Subacute petrositis with abducent paralysis is a rare complication of bacterial otitis. The conditions necessary for

its occurrence are extremely limited. There must be an unobstructed pathway through at least one chain of air cells into the petrous bone. The bacteria must be of sufficient virulency or the host must have a sufficiently low resistance to the attacking organisms. The children involved are mostly unhealthy and below par. Dorello's canal must be small enough so that a slight swelling of its wall is sufficient to produce paralysis.

2. The pain is caused by stretching of the dura, due to its inflammatory edema, on the anterior or posterior surface of the petrous pyramid, thus stimulating the rich terminal network of nerve fibres at their sensory endings in the dura. The pain is reflected and felt at the more sensitive areas supplied by these nerves.

3. The diagnosis of the condition is vital.

Petrosal osteitis and osteomyelitis must be recognized and differentiated. If these two conditions are reliably excluded, the mortality should be extremely low, much below 7 per cent (Gradenigo's estimate in 1908, and Perkins'[21] series with 11.5 per cent mortality, reported in 1910).

BIBLIOGRAPHY.

1. Dorello in 1905 suggested that compression in this canal produced paralysis of the VIth nerve.
2. Pragier: *Monatsschr. f. Ohrenheilk.*, Dec., 1933.
3. Vail, H. H.: *Arch. Otolaryngol.*, May, 1936.
4. Tremble and Pennfield: *Arch. Otolaryngol.*, May, 1936, p. 573.
5. Seydell, E. M.: *Arch. Otolaryngol.*, Oct., 1935.
6. Head: *Brain*, 1893.
7. Myerson: *Arch. Otolaryngol.*, July, 1935.
8. Friesner, I.: *Arch. Otolaryngol.*, Dec., 1935.
9. Roberts: The Laryngoscope, April, 1934.
10. Profant, H. J.: *Arch. Otolaryngol.*, March, 1931.
11. Houser, K. M.: *Arch. Otolaryngol.*, Oct., 1932.
12. Wilson, Gordon: *Ann. Otol., Rhinol. and Laryngol.*, 1939.
13. Friesner, I.: Arch. Otolaryngol., Oct., 1935.
14. Guild: *Arch. Otolaryngol.*, Oct., 1935.
15. Jones, Marvin: *Arch. Otolaryngol.*, Oct., 1935.
16. Wilson, Gordon: *Arch. Otolaryngol.*, Oct., 1935.
17. Eagleton, Wells P.: *Arch. Otolaryngol.*, 1935.
18. Perkins, J. A.: *Ann. Otol., Rhinol. and Laryngol.*, Sept., 1910.
19. Friesner, I.: *Arch. Otolaryngol.*, Dec., 1935.
20. Lillie: *Arch. Otolaryngol.*, Oct., 1935.
21. Perkins, Chas. E.: *Ann. Otol., Rhinol. and Laryngol.*, Sept., 1910.

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THE DEVELOPMENT OF THE AUDIOMETER.*†

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The modern audiometer is the result of the evolutionary demands of otologists, acoustic engineers and others who desired to learn more about the nature of the hearing process. The student who is interested in tracing its development finds himself confused, in the foreign journals at least, with the somewhat promiscuous use of the words, acumeter, acoumeter, audimeter, audiometer and sonometer. This should be expected since these words were coined from Greek or Latin roots having almost identical meanings. Dorland[14] defines the audiometer as "a device to test the power of hearing," an acoumeter as "an instrument for use in testing the accuracy or acuteness of the hearing," and a sonometer as "an apparatus for testing acuteness of hearing." In none of these definitions is there any specification as to the nature of the source of the sound stimulus. The German word "Hörmesser" might with equal accuracy be translated into the English language as an acumeter, audiometer or sonometer. Politzer's Hörmesser,[44] described in 1877, in which the sound stimulus resulted from the noise of a small weight falling upon a rigid bar, in the English translation of his text is called an acoumeter; Urbantschitsch's "Elektrischer Hörprüfungs Apparat"[53] is called an electric acoumeter in Dench's American text;[13] a series of bells with automatic striker, designed for hearing tests by Beerwald was called an acoumeter by Hartmann and Schulte[27] in their review of the literature for the year 1887; and Ostmann's[42] series of tuning forks was called an "objective audiometer." As late as 1937, the Council on Physical Therapy of the American Medical Association gave this definition:[47] "A clinical audiometer is an instrument for measuring the acuity and range of hearing." Regardless of this confusion of nomenclature and definition, the word audiometer today among otologists and acoustic engineers is quite generally restricted to instruments in which the stimulus

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tones are generated electrically. This being the case, the invention of the telephone by Alexander Graham Bell in 1876 places a definite time limit upon the search for the origins of the modern audiometer. Changes in design have rapidly followed the evolutionary development of the knowledge of the applications of the electric current in telephony.

Credit for the earliest application of the electric current to hearing tests undoubtedly rests between two investigators, A. Hartmann and D. E. Hughes. Hartmann,[24] in the early part of 1878, devised an instrument using an electric current with a telephone receiver for hearing tests. This instrument is called an "acoumeter" in the English translation of his text.[25] He describes it as follows: "After the invention of the telephone, the author endeavored to obtain an exact graduation of sound by means of electric currents. In the circuit is placed *1.* a tuning fork, by which the current is interrupted at regular intervals; *2.* a rheocord, or a sliding induction apparatus, by means of which the intensity of the current can be varied and exactly regulated at will; and *3.* a telephone, at which is heard a tone corresponding with that of the vibrating tuning fork, of more or less intensity according to the strength of the current. Although the hearing-test can be made easily and rapidly by means of such an apparatus, it is, unfortunately, somewhat too complicated, and as only a small number of tones can be produced, the apparatus has not yet been introduced into practice." Neither the original article nor his text give an illustraton of this instrument. From his statements it is evident that it did not receive great consideration even from the inventor during the time between 1878 and 1887. Hartmann was apparently not satisfied with it, for he stated in his text,[26] "A perfect acoumeter must possess the following qualifications. It must have as great a range of tones as possible, which can always be produced at the same intensity; it must be handy and simple, so that it can be employed without difficulty in all ordinary examinations. It must further be so constructed that it cannot only be used for testing air-conduction but also the bone-conduction."

Fig. 1 shows the electrical circuit used in Hartmann's acoumeter as it was modified by Korting.[36] The tuning fork for interrupting the primary circuit described by Hartmann is

not shown in the diagram. Korting states that it was used for testing the hearing of army recruits.

In 1879, Hughes[31] described an instrument called an "induction balance," which he used for the analysis of certain metals. He states that he ". . . joined to this instrument an

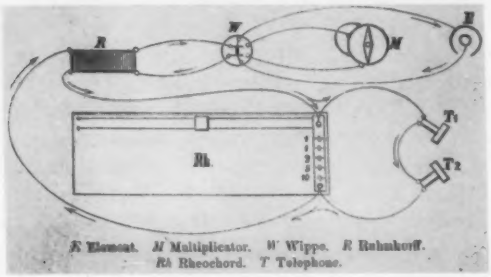

Fig. 1. Wiring diagram of Korting's (1879) modification of Hartmann's audiometer of 1878. The essentials are the battery (E), induction coil (R), sliding inductance (Rh) and telephone receivers (T). The tuning fork described by Hartmann is not shown.

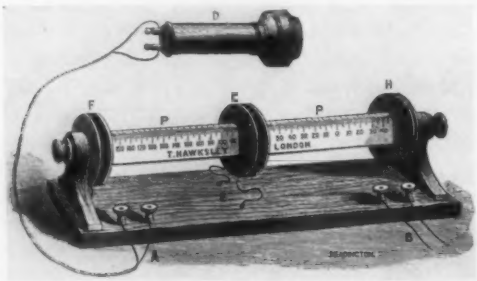

Fig. 2. Illustration from the 1893 edition of Delby's text showing the audiometer designed by Hughes (1879). Batteries and tuning fork interrupter are attached at (B). No tone is produced when the movable coil (C) is midway between the fixed coils (F) and (H). As (C) is moved along the bar (P) toward either fixed coil, the tone in the telephone receiver increases. Intensity values were in terms of distance from the center.

instrument to which I have given the name of electric sonometer." During the same year, Richardson[49] wrote quite enthusiastically, ". . . the world of science in general, and the world of medicine in particular, is under a deep debt of gratitude to Prof. Hughes for his simple and beautiful instrument which I have christened the audimeter, or less accurately but more euphoniously, the audiometer."

This audiometer must have been used by certain otologists other than Richardson, for the illustration shown in Fig. 2 was taken from Dalby's text of 1893. J. Orne Green[22] exhibited this instrument in America and reported that he "was not prepared to give his opinion of the instrument in full at this time, but could say that it was not an accurate measure of the hearing power in general as in relation to talking, but only in relation to this special kind of sound, which is a different matter, the hearing for one kind of sound being often impaired without that for another suffering any change."

Politzer was at this time apparently quite optimistic about these instruments, for in his text[46] he states, "In the Physiological Society of Berlin (Discussions of Jan. 11, 1878), Arthur Hartmann demonstrated a new method of testing the hearing. It is possible to graduate a sound created in a telephone with an exactitude hitherto unapproached, as the electric flashes transmitted to the telephone can be altered in a precisely definable manner by the interpolation of different kinds of resistance. It may be expected that many questions regarding the perceptions of the ear, which are still unexplained, will be brought nearer to solution by this important discovery."

Both of these instruments apparently have similar electrical circuits. The current in the primary of an induction coil is interrupted by an electric tuning fork. The interruptions induce an alternating current in the secondary circuit which includes the telephone receiver. The pitch of the fundamental tone in the receiver is determined by the rate of vibration of the tuning fork. Different fundamental tones can be secured only by installing forks of different pitch. This tends to make the apparatus complicated, unstable, cumbersome and difficult to standardize. It is extremely difficult to operate electric tuning forks with frequencies greater than 1,000 vibrations per second. This limits the tones produced to those of rather low pitch. More than this, a pure tone cannot ordinarily be produced in a receiver in the secondary of this type of circuit.

Two men in 1882 described new electrical instruments devised for the purpose of testing hearing, Ladriet de Lacharriére[39] and Boudet de Paris.[43] Descriptions of these instruments in the sources available for this study are mea-

ger. The audiometer of Ladriet de Lacharriére is illustrated in Fig. 3. The circuit is apparently similar to that of the sonometer developed by Hughes. The sliding induction coil is replaced by those of fixed type. A single electrical tuning fork is an integral part of the set and the batteries are enclosed. The compactness of the set leads one to think that an attempt was made to produce it commercially but no evidence was found that it was used by contemporary otologists.

No illustration of the complete audiometer produced by Boudet de Paris could be found. Fig. 4 shows only the wiring

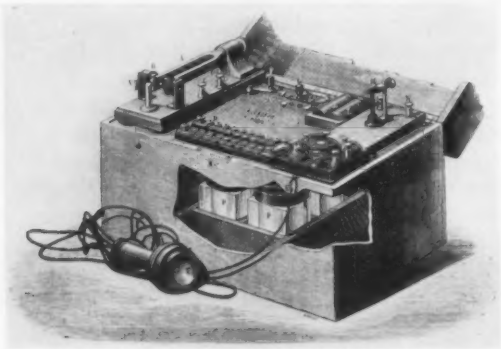

Fig. 3. Audiometer of Ladriet de Lacharriere (1882). The compact appearance of the instrument leads one to think that commercial production may have been attempted.

diagram, which appears to be a modified form of a Wheatstone's bridge. The symbols used by the electrical engineers of 1884 were different from those in use today, so in the absence of a detailed description it is not possible to state the exact nature of its circuit. No electric tuning fork is shown, so the nature of the sound stimulus in the telephone receiver cannot be determined. The set shows a galvanometer, which may indicate an attempt at more accurate current control. Baratoux[3] used this instrument and apparently modified it in certain respects, for Hartmann wrote concerning it, "Baratoux describes the audiometer previously employed and mentions Boudet's as he has modified it. He retains the rheostat and telephone but omits the microphone. An electric tuning fork serves to interrupt the current."

Urbantschitsch's text[53] of 1884 shows a picture of his "Elektrischer Hörprüfungs Apparat." This apparatus became sufficiently well known that it was described as an "electric acoumeter" in the 1909 edition of Dench's[13] text, although no records of its clinical use were given. From the illustration

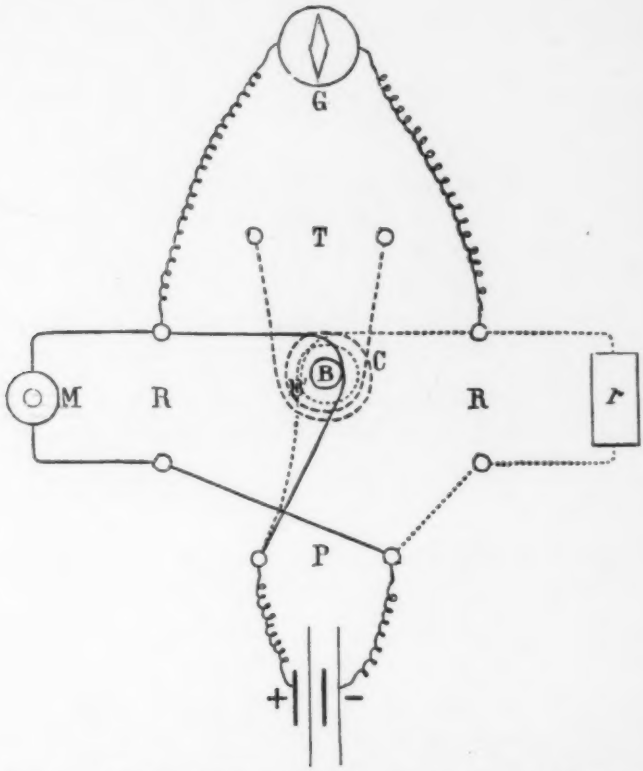

Fig. 4. Wiring diagram of the audiometer of Boudet de Paris (1882). A modern electrical engineer would have difficulty in determining the essential parts. It appears that two telephone receivers were employed. How the alternating current was produced is not clear.

in Fig. 5, it appears to have been constructed on the same principle as the sonometer of Hughes. The current from a battery, interrupted by an electric tuning fork or Neef's hammer, passes through two identical primary coils mounted and conveniently spaced on a rigid bar. A third coil, connected to

the telephone receiver, slides on the rigid bar toward either primary. The induced alternating current in the secondary is nil when it is midway between the primary coils. As it approaches either primary, the induced current increases proportionately.

During the next three decades other writers described instruments for hearing tests which used the induction coil

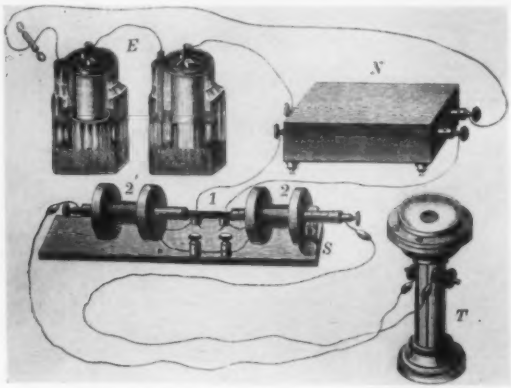

Fig. 5. A Vienna contribution to audiometry. Urbantschitsch's "Electrischer Horprufungs Apparat" of 1884. The tuning fork has been replaced by a buzzer circuit breaker in the primary circuit. Dench's 1909 text called this an "electric acoumeter."

to transform an interrupted direct current into an alternating current. Among these should be mentioned Cozzolino[11] (1885), Cheval[10] (1890), Jacobsen[32] (1895), Seashore[50] (1899), Trétop[52] (1908), Gradenigo[21] (1907) and Foy[19] (1916).

In Seashore's audiometer, shown in Fig. 6, the secondary windings were arranged as a series of coils in which the number of turns varied in a logarithmic ratio. This gave variations in the loudness of the stimulus to correspond with the Weber-Fechner law. Seashore claims to have coined the word "audiometer" but we have seen that it was used earlier by Richardson and others. McMillan used this instrument in tests of children in the Chicago Public Schools. No other clinical reports are available.

It became evident with increasing knowledge of auditory function that hearing tests using a simple impure tone gave

very little information of clinical value. Politzer[44] criticized instruments of this type as follows: "The acoumeter of Hartmann, Dalby, Cozzolino, Gradenigo and Urbantschitsch, made after the principle of the sonometer of Hughes, in which the striking of a Neef's hammer is conveyed to the ear by a telephone and by means of changing the induction coil, gradually made weaker and stronger, I have not tested in practice . . .

Fig. 6. The American influence. Seashore's audiometer (1899). The tuning fork is not shown. A series of inductances is enclosed. Intensities are regulated according to the Eeber-Fechner law.

it possesses, as all acoumeters do, the fault that we cannot judge, from the increase of the hearing distance for the tone of the acoumeter, as to the increase of the hearing distance for speech." Bryant,[7] too, stated, "The objections to the use of electricity in testing are the bulkiness of the apparatus, the amount of care required to keep it in running order and, lastly, the expense."

Bezold,[5] in 1898, realized the necessity for otologists to use a wide range of tones in hearing tests if they were to be of diagnostic value and presented his "continuous tone series"

of tuning forks. He pointed out[6] the fact that tones and noises were different manifestations of the same physical phenomenon and said, "We have, therefore, completely examined the hearing, as soon as we know quantitatively the hearing for all tones, and we do not need to care for the noises."[4] He also stated, "entire series of tones were of no use to us

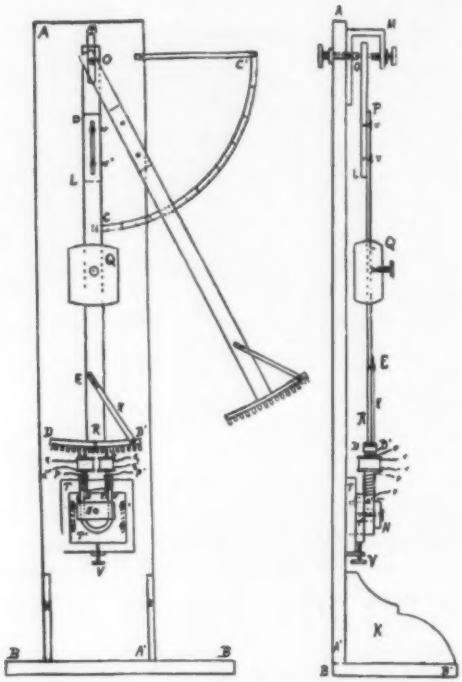

Fig. 7. The pendulum generator of Stefanini (1914). Current is generated as the toothed pendulum falls above a permanent magnet. The pitch of the tone is governed by the speed of the pendulum as it falls.

until we ceased to think of noises as different from impressions of sound. The separation of noises from tones long hindered the development of otology because it gave rise to the idea of a separate region for the perception of noises outside the cochlea, in another portion of the labyrinth." Ostmann[42] also reported in the same critical manner, "We are hindered by imperfections of our apparatus so that we

have not yet obtained a sufficiently broad foundation for a perfect audiometer."

It remained for Stefanini[21] to construct, in 1914, an electric generator which would produce an alternating current with a range of frequencies which could be conducted to a telephone receiver for use in hearing tests. This generator is shown in Fig. 7. The pendulum carries a row of iron teeth which, as the pendulum falls, pass through an electromagnetic field, causing magnetic changes. This induces an alternating current in the generator, the frequency depending upon the speed with which the teeth pass through the field. This is governed by the distance which the pendulum falls. The number of teeth on the pendulum was small and the duration of the stimulus tone was necessarily quite short. No results of the clinical application of the tests with the instrument were given.

Alexanderson[1] and Duddell[15] devised somewhat similar generators for radio communication in which the teeth were an integral part of a wheel which rotated in a magnetic field. Cahill[9] had a similar generator, or rather a series of generators, by means of which he proposed to produce and transmit music by wire. None of these generators were used for hearing tests.

Dean and Bunch[12] saw the possible application of this type of generator for hearing tests and in 1919 presented their "pitch range audiometer." This instrument consisted essentially of a small alternating current generator driven by a variable speed motor, so that tones between 30 to 10,000 cyles were produced. They claimed for this instrument that: "*1.* It produces each and every tone within the significant range of tonal hearing without any gap whatsoever. *2.* It produces a relatively pure tone, pure enough for all practical purposes; and we have at our command the mechanical principles which determine the form of the sound wave. *3.* It furnishes us a convenient method of varying the intensity from below the threshold up to a sound that is on the verge of being painful for any pitch. *4.* It enables us to record and measure instantly and with precision, and at the time of hearing, both the pitch and the intensity of the tone. *5.* It enables us to sweep through the entire range of pitches at

one stroke for any steps controlling intensity and to sweep through at a single stroke all intensities for a given pitch. *6.* The operation of the instrument is so simple and quick that a complete measurement can be made in a very small fraction of the time employed in making the ordinary tests now in vogue with otologists." The instrument was provided with a key by means of which the stimulus could be interrupted, and a signal light and key for indicating the response of the patient.

The motor-generator and control panel of this audiometer are shown in Fig. 8. Charts secured by means of tests with

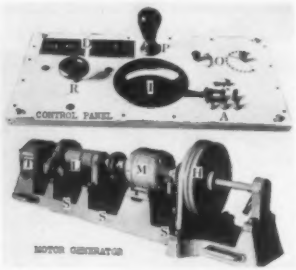

Fig. 8. The motor-generator and control panel of the pitch range audiometer of Dean and Bunch (1919). The pitch of the stimulus is governed by the speed of the motor (M) which turns two generators (H) and (L). The intensity was regulated by resistances at (O). The pitch is read on the dial at (I) which is connected to the tachometer (T).

it were called "hearing fields." Since it was the usual procedure to test for all tones, "complete" and "partial" gaps in the tonal range were found rather frequently. Prior to 1927 these writers published several articles relating to the application of audiometric tests to clinical otology and attempted to interpret the results secured. All of these appeared in the English and American journals. The pitch range audiometer was not produced commercially.

Concerning this audiometer, Kerrison[34] wrote, "An ingenious mechanism which, if it could be made generally available, would add greatly to the accuracy and scientific value of our hearing tests is the so-called tone-range audiometer devised by Dr. Lee Wallace Dean and Mr. C. C. Bunch, of the University of Iowa. . . . The advantages of such an instrument over tuning forks in the detection of gaps in tone perception are obvious. As with every notable advance in diagnostic

method and accuracy, it is probable that this mechanism in the hands of its distinguished inventors will add to our scientific knowledge of disease by establishing shades of functional difference between certain closely related lesions — shades of difference not definitely determinable by the older methods."

Concerning its development and use, Macfarlan[40] states, "From this time dates the development of the scientific work in testing the hearing across the range of frequencies."

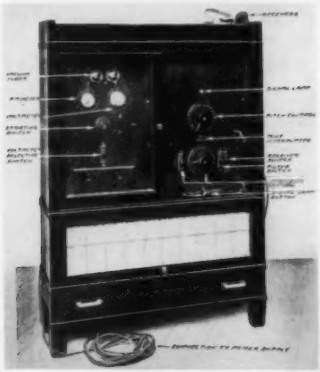

Fig. 9. The first commercially produced audiometer of the vacuum-tube type. The 1A audiometer of the Western Electric Co. Fletcher and Wegel. 1922.

The next step in the development of the modern audiometer followed the rather general application of the vacuum tube in radiotelephony. It is not within the scope of this paper to discuss the theory or history of vacuum tubes. It is sufficient to state that radio engineers have discovered that by their use oscillating electric currents of almost any desired frequency can be obtained. Taking advantage of this for otology, Minton and Wilson[41] described a vacuum tube set which was designed and used for the purpose of testing hearing. During the same year, Guttman[23] also presented a vacuum tube set which he used for hearing tests, which he called an "acumeter." Neither of these sets was commercialized and studies of their application appeared only under the names of these investigators.

The engineers of the American Telephone and Telegraph Co. and its subsidiaries, the Bell Telephone Laboratories and

the Western Electric Co., had, through their interest in telephonic communication, for some time been doing considerable research in the problems of the frequency characteristics of speech[16] and the sensitivity of the ear.[17] In 1922, Fowler and Wegel[18] presented to the otological world the Western Electric 1A Audiometer which had been developed by these engineers. This the first audiometer of the vacuum tube type to be produced commercially, is illustrated in Fig. 9.

The charts and hearing curves were called "audiograms," an audiogram being defined as "a curve plotted so as to show the variation of minimum audible sensitivity with frequency where the stimulus is a pure tone." They describe the essential parts of the audiometer as follows: "The oscillator circuit is capable of furnishing a very pure tone without wave filters, since particular care was taken in the selection of the circuit, coils and condensers. This is very important because throughout the lower portion of the frequency range, in particular, the harmonics of an impure tone are usually more audible than the fundamental tone itself, so that the reading of minimum audibility depends on the sensitivity of the ear to the overtones and not to the fundamental.

". . . The attenuator used is direct reading in logarithmic units and if the logarithmic relation between stimulus and sensation be assumed, it may be considered to read directly in units of sensation rather than stimulus. This attenuator is a low resistance iterated network developed from the point of view of avoiding interference between its input and output ends.

". . . The receiver is one in which the diaphragm is damped by air damping to such a degree that the effects on the diaphragm, of placing it on the ear, is negligible."

The Western Electric Co. was for a number of years the only one producing audiometers on a commercial scale. The almost prohibitive price of the 1A audiometer — approximately $1,500.00 — was a primary factor which prevented its more general use. A less expensive model, the 2A, which used as stimulus tones only the octaves of *c* from 64 to 8,192 cycles, followed the 1A and was used by a considerable number of otologists. The 3A, in which the stimulus was a complex noise, not unlike that produced by Urbantschitsch's model

mentioned above, was offered for rough hearing tests in commercial plants, but found little favor with otologists who wished pure tones as stimuli.

In England. also in 1922, Hastings and Tucker[28] described a vacuum tube set used for hearing tests and listed the characteristics which they considered desirable in such an instrument. No illustration accompanied this article and it seems probable that it did not reach a production basis.

The 4A audiometer of the Western Electric Co., a later model of which is shown in Fig. 10, was constructed along

Fig. 10. The No. 4A audiometer and a No. 4A receiver holder, showing the eight compartments and the manner of nesting telephone headsets.

different principles. Instead of using tones produced by a vacuum tube circuit, this instrument is constructed on the principle of the phonograph in which the stimulus sounds are spoken numbers. Bryant[8] and others had attempted to use the phonograph for hearing tests prior to this time, but the variation in the surface noises and inability to standardize the stimulus sounds in the phonographs employing the old mechanical reproducers prevented its universal use. The development of the new electromagnetic pickup made it possible to minimize the defects of the older type and to use the phonograph in hearing tests. The numbers were spoken so that they are cut in the phonograph disc and reproduced through the telephone receivers at fixed intensities. As the test proceeds, the intensity of the voice decreases in gradu-

ated steps until the threshold of audibility is reached. The instrument can be used for either group or individual tests, 40 receivers being provided with each phonograph when desired, so that an equal number of listeners may be tested at the same time. A convenient chart is provided, on which the listener writes the numbers as he hears them. This limits its use to those who understand the spoken English language. It cannot be used in group tests of children who cannot write or who are too immature to record the numbers at the speed with which the words are reproduced. In individual tests the listener may speak the words as he hears them if he is unable to write.

Knudson,[35] who for a number of years had been interested in problems of hearing, co-operated with Jones[33] and in 1924 presented a vacuum tube set designed for hearing tests, which they called an "audioamplifier." This instrument was later modified and produced commercially by the Sonotone Corp. under the name of the Sonotone Jones-Knudson Model 1 Audiometer.

Kranz,[37] who also for a number of years had been interested in problems of hearing, presented an audiometer[38] in 1924. In one of his earlier studies he used a thermophone in place of the telephone in the vacuum tube circuit. This writer, in co-operation with A. G. Pohlman, presented several articles on the clinical application of hearing tests.

Within the past few years several other audiometers have been offered commercially, some of which have been described in the otological literature. One difficulty in securing the general approval of otologists has been due to the fact that each manufacturer calibrated his product according to his own standards. The standards of different manufacturers did not always agree. This made it impossible to secure identical records on the same patient in tests with different audiometers and has led to considerable confusion. Hayden[29] in 1938, after testing several patients with four different makes of audiometers, reported that the records showed considerable variation due to the fact that the intensity calibration of the audiometers did not correspond. Regardless of this objectionable feature, he stated, "Excellent features were found to be common to all. . . With any of the instruments,

hearing can be measured more accurately and rapidly than with any previous methods or instruments. The audiogram is the best means of recording hearing loss for diagnosis of otologic disease, fitting of hearing aids and for medicolegal purposes."

Otologists, however, have demanded that audiometers be standardized, both as to frequency and intensity. In order to comply with this demand the Council on Physical Therapy of the American Medical Association requested a number of otologists to serve on a committee to set up certain standards which must be met in order to secure the acceptance of the Council for that product. Among the requirements set up by the Council[48] on the recommendation of this committee were those dealing with the number of tones produced, their purity, intensity values, maximum output for the various tones, maximum intensity, etc., as well as the limits of variation from those set values which may not be exceeded. These were minimum requirements. Some of the audiometers now accepted present additional features which were not required. For example, they may offer higher and lower tones than specified or have a greater acoustic output. One accepted audiometer provides a sweep of all frequencies in addition to being calibrated for the specified octave tones. The tendency of the manufacturers has been to produce an instrument which will meet the needs of the most exacting otologist.

At the present time, three audiometers have been accepted by the Council, the Sonotone Jones-Knudson Model 1, the Maico Model D5 and the Western Electric 6B. Others have been submitted and are undergoing consideration.

A brief search through the literature is sufficient to convince one that the greater part of the present day studies in audiometry originated in America. However, several reports have recently come from England. One outstanding study by Bárány[2] has recently come from Sweden. He states, "In Europe, electrical audiometers and bone conduction receivers have not yet come into general use even as research tools, and only a few nonclinical papers using them for bone conduction work have been published." Another study, also from Sweden, by Holmgren[30] appeared recently. He presented an audiometer which produced all tones from 62 to 15,000 cycles,

reading in 1 dcb. steps from the threshold to 125 dcb. With existing conditions in Europe, it cannot be expected that scientific studies of this type will be forthcoming from these countries for some time. A careful perusal of the American journals will probably keep the student informed of the progress in this field.

BIBLIOGRAPHY.

1. Alexanderson, E. F. W.: High Frequency Generator. *Proc. Am. Inst. Elec. Engin.*, *28*:399, 1909.

2. Barany, E.: A Contribution to the Physiology of Bone Conduction. *Acta Oto-Laryngol. Supp.*, Vol. 26, Uppsala, 1938.

3. Baratoux, J.: Audiometers. *Rev. Mens. de laryngol. d'tol.*, No. 8, 1882. See also review by Hartmann, A.: Progress of Otology. *Arch. Otol.*, 12:168, 1883.

4. Bezold, F.: On the Present State of the Various Tests for Hearing. *Arch. Otol.*, *25*:275, 1896.

5. Bezold, F.: Six Additional Cases of Necrosis of the Labyrinth. Trans. by Dr. Arnold Knapp. *Arch. Otol.*, *27*:158, 1898.

6. Bezold, F.: Textbook of Otology. Trans. by J. Holinger. E. H. Colegrove Co., 1908, p. 56.

7. Bryant, W. S.: A System of Diseases of the Ear, Nose and Throat. Edited by C. H. Burnett. Vol. 1, p. 98, J. B. Lippincott Co., Philadelphia, 1893.

8. Bryant, W. S.: A Phonographic Acoumeter. *Arch Otol.*, 33:438, 1904.

9. Cahill, T.: Electrical Music. *Scien. Am.*, 94:268, 1906.

10. Cheval, V.: Electroacoumetére; nouvelle methode pour la recherche de la surdité dans les Consuls de milice et de revision, etc. *Bull. de L'acad. Roy. de Méd. de Belgique*, 4:258, 1890.

11. Cozzolino, V.: L'Audiometro Elettro-Telefonica. *Boll. d. Mallattie d. 'Orec., d. Gola e d. Naso*, 3:5:85, 1885.

12. Dean, L. W., and Bunch, C. C.: The Use of the Pitch Range Audiometer in Otology. The Laryngoscope, 29:453, 1919.

13. Dench, E. B.: Diseases of the Ear. Fourth Ed. D. Appelton and Co., New York, 1909, p. 146.

14. Dorland, W. A. N.: The American Illustrated Medical Dictionary. W. B. Saunders Co., Philadelphia, 1922.

15. Duddell, V.: High Frequency Alternator. *Proc. Phys. Soc. of London*, 19:431, 1903-1905.

16. Fletcher, H.: The Nature of Speech and Its Interpretation. *Jour.* Franklin Inst., 193:729, 1922.

17. Fletcher, H., and Wegel, R. L.: The Frequency Sensitivity of Normal Ears. *Phys. Rev.*, 19:553, June, 1922.

18. Fowler, E. P., and Wegel, R. L.: Audiometric Methods and Their Applications. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, p. 96, 1922.

19. Foy, R.: Examen des surdités de guerre. *Proc. Nouv. d'Acoumétrie. La Presse Med.*, 24:595, 1916.

20. GRADENIGO, G.: Acoumetre Telephonique du Prof. Stefanini. *Arch Int. d. Laryngol.*, etc., 24:500, 1907.

21. GRADENIGO, G.: Suggestions in Audiometry. THE LARYNGOSCOPE, 23:770, 1913.

22. GREEN, J. O.: Repart by James J. Putman, Secy., Boston Society of Medical Sciences. *Boston Med. and Surg. Jour.*, 103:474, 1880.

23. GUTTMAN, J.: A New Method of Measuring Hearing Power by Means of an Electric Acumeter. THE LARYNGOSCOPE, 31:960, 1921.

24. HARTMANN, A.: Eine neue Methode der Hörprüfung mit Hülfe elektrischer Ströme. *Arch. f. Physiol.*, p. 155, 1878.

25. HARTMANN, A.: The Diseases of the Ear and Their Treatment. Trans. from the 3rd German Ed. by James Erskine. G. P. Putnam's Sons, New York, 1887, p. 26.

26. HARTMANN, A.: *Ibid.*, p. 25.

27. HARTMANN, A., and SCHULTE, E.: Progress of Otology. *Arch. Otol.*, 16:49, 1887.

28. HASTINGS, S., and TUCKER, W. S.: An Attempt to Standardize Tests for Hearing. *Proc. Roy. Soc. Med.*, Part 3, Sect. Otology, 16:1, 1922-1923.

29. HAYDEN, A. A.: Hearing Aids from Otologist's Audiograms. *Jour. A. M. A.*, 111:592, 1938.

30. HOLMGREN, L.: Hearing Tests and Hearing Aids. *Acta Oto-Laryngol. Supp.*, Vol. 34, 1939.

31. HUGHES, D. E.: On an Induction-Currents Balance and Experimental Researches Made Therewith. *Proc. Roy. Soc.*, 29:56, 1879.

32. JACOBSEN, L.: Ein neuer telephonischer apparat zur Untersuchung und Behandlung des Gehörogans. *Deut. Med. Wchnschr.*, 11:908, 1885.

33. JONES, I. H., and KNUDSON, V. O.: Functional Tests of Hearing. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, p. 120, 1924.

34. KERRISON, P. D.: Diseases of the Ear. 1930 Ed. J. B. Lippincott Co., Philadelphia, p. 87.

35. KNUDSON, V. O.: Sensibility of the Ear to Small Differences in Intensity and Frequency. *Phys. Rev.*, 19:261, 1922.

36. KORTING: Ueber telephonische Hörprüfung. *Deut. Mil. Ztschr.* 7:337, 1879.

37. KRANZ, F. W.: Minimum Sound Energy for Audition. *Phys. Rev.*, 18:384, 1921.

38. KRANZ, F. W.: Some Aspects of the Problem of the Testing of Audition, with Demonstration of a New Portable Apparatus. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, p. 140, 1924.

39. LACHARRIERE, L.: Des Moyens d'apprécier la sensibilité auditive—Nouvel Audiométre. *Ann. des Mal. d. l'Orielle*, etc., 8:134, 1882.

40. MACFARLAN, D.: History of Audiometry. *Arch. Otolaryngol.*, 29:5.4, 1939.

41. MINTON, J. P., and WILSON, J. G.: The Sensitivity of Normal and Defective Ears for Tones at Various Frequencies. *Proc. Inst. Med.*, 3:157, 1921.

42. OSTMANN: Nature and Aim of Obiective Measurement of Hearing, and on the Use of the Objective Audiometer. *Arch. Otol.*, 24:267, 1905.

43. Paris, B.: Détermination de la sensibilitié auditive. *Ann. des Mal. d. l'Orielle*, 8:301, 1882.

44. Politzer, A.: Ueber einen einheitlichen Hörmesser. *Arch. f. Ohrenheilk.*, 12:104, 1877. See also abstract by Blake C.: *Arch. Otol.*, 6:336, 1887.

45. Politzer, A.: Diseases of the Ear. Trans. from the 3rd German Ed. by Oscar Dodd. Lea Bros. and Co., Philadelphia, 1894, p. 131.

46. Politzer, A.: Diseases of the Ear. Trans. by J. P. Cassells. Henry C. Lea's Sons and Co., Philadelphia, 1883, p. 188.

47. Report of the Council on Physical Therapy. Tentative Minimum Requirements for Acceptable Audiometers. *Jour. A. M. A.*, 109:1812, 1937.

48. Report of the Council on Physical Therapy. Minimum Requirements for Acceptable Audiometers. *Jour. A. M. A.*, 113:732, 1939.

49. Richardson, B. W.: Some Researches with Prof. Hughes' New Instrument for the Measurement of Hearing; the Audiometer. *Proc. Roy. Soc. Med.*, 29:65, 1879.

50. Seashore, C. E.: An Audiometer. *Univ. of Iowa Studies in Psychology*, 2:158, 1899.

51. Stefanini, A.: Alternatore Pendolare Electromagnetico. Il Nuovo Cimento. Serie VI, 7:261, 1914.

52. Tretop, d'Anvers: Acoumétrie Millimetric. *Arch. Intern. de Laryngol.*, etc., 26:157, 1908.

53. Urbantschitsch, V.: Lehrbuch der Ohrenheilkunde. Urban u. Schwartzenburg, 1884, p. 409.

54. Wilson, J. G., and Minton, J. P.: The Sensitivity of Normal and Defective Ears or Tones of Various Frequencies. *Proc. Inst. of Med.*, 3:157, 1921.

PONTOFACIAL ANGLE TUMORS WITH PARTICULAR REFERENCE TO THE INVOLVEMENT OF THE ACOUSTIC NERVE.*

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A discussion of tumors of the acoustic nerve makes it desirable to review the better known anatomical and clinical features of tumors of this type and in this location, and to follow it by a survey of the clinical and pathological material such as would emphasize the more typical manifestations of the acoustic neuroma and, finally, to contrast them with those which characterize other tumors in the pontofacial angle. In this manner it may be possible to bring to the fore some instructive data in differential diagnosis of expanding lesion *in* and *about* this area. In my remarks I shall often make use of the term *pontofacial angle tumor* in preference to that of acoustic neuroma, and because of this it will be advantageous to recall some of the more significant features of this anatomical landmark.

The pontofacial angle (see Fig. 1) is that region at the base of the brain which is bounded anteriorly by the posterior border of the pons, and medially and posteriorly by the lateral aspect of the medulla oblongata, and laterally by the posterior border of the ipsilateral cerebellar hemisphere. Thus, because it is formed by the apposition of these three structures, it is often and quite correctly referred to as the pontomedullocerebellar angle. With the *facial* nerve taking its superficial origin in that region, it has been often referred to as the pontofacial angle. Within this angle, in addition to the facial nerve, is the entrance of the *cochlear* and *vestibular* divisions of the *acoustic nerve*, and what is also quite significant, just a short distance rostrad to these nerves at the side of the pons, there is the exit of the motor and the entrance of the sensory fibres of the *trigeminal nerve;* somewhat caudad and on the side of the medulla are the rootlets of the *glossopharyngeal* and *vagus nerves.* Here, thus, is a rather

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crowded territory and it is hardly necessary to stress the significance of the structures about it, such as the cerebellum, the great equilibrator and synergizer of muscular activity; the pons, the seat of both sensory and motor nuclei of the trigeminal nerve; the pontofacial angle, the site of superficial origin of the VIIth and VIIIth nerves; and, finally, the lateral region of the medulla with the fibres of the glosso-

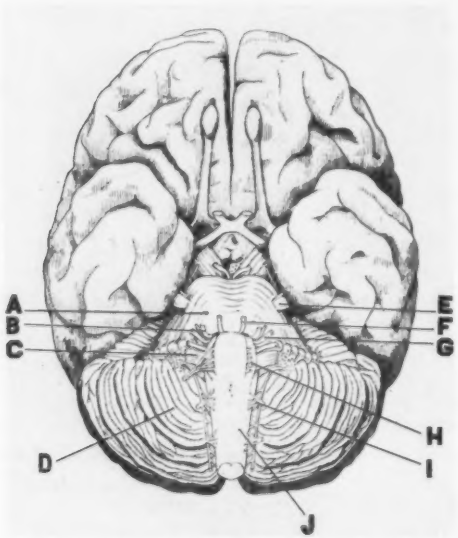

Fig. 1. Base of brain, illustrating structures in and about the pontofacial angle (drawing). (A) Pons. (B) Facial nerve. (C) Glossopharyngeal and vagus nerves. (D) Cerebellum. (E) Trigeminal nerve. (F) Abducens nerve. (G) Acoustic nerve. (H) Hypoglossal nerve. (I) Spinal accessory nerve. (J) Medulla oblongata.

pharyngeal and vagus. No less significant is the fact that within the substance of the pons and the medulla, within the depth of these two subdivisions of the brain stem, are important pathways conveying impulses up and down this rather busy roadway (see Fig. 2). Here are the ascending *spinocerebellar tracts*, ventral and dorsal, carrying muscle, joint and tendon sense from the spinal cord to the cerebellum; the descending *pyramidal tract fibres*, which carry motor impulses from the cerebral cortex to the ventral horn cells in the spinal cord; and the exceedingly significant *posterior longitudinal bundle*, providing a medium for the oculovestibulo-

spinal mechanism. Moreover, within the pons there are the extensions of the *cochlear* pathways, such as the *trapezoid* body with the *lateral lemniscus*, as well as the *nuclear* masses of the vestibular branch of the VIIIth nerve. It is quite obvious that the functions of all these structures situated within or adjacent to this angle are likely to be affected by a neighboring compressing agent and give rise to signs and symptoms of localizing value.

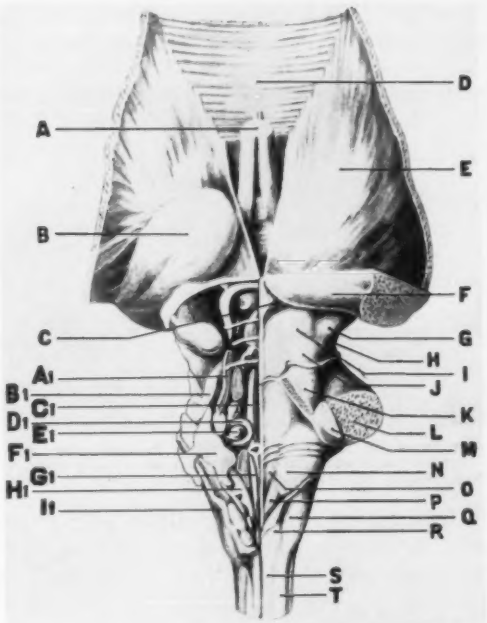

Fig. 2. The internal structures of the brain stem, with particular emphasis on those in the proximity of the pontofacial angle (drawing). (A) Fornix. (B) Thalamus. (C) Pineal body. (D) Corpus callosum. (E) Corona radiata. (F) Pulvinar. (G) Medial geniculate body. (H) Posterior quadrigeminal brachium. (I) Anterior quadrigeminal body. (J) Posterior quadrigeminal body. (K) Brachium conjunctivum. (L) Brachium pontis. (M) Restiform body. (N) Area acustica. (O) Hypoglossus trigone. (P) Vagus trigone. (Q) Cuneate tubercle. (R) Clava. (S) Funiculus gracilis. (T) Funiculus cuneatus. (A1) Mesencephalic root of trigeminal nerve. (B1) Brachium conjunctivum. (C1) Medial longitudinal bundle. (D1) Genu of facial nerve. (E1) Nucleus of abducens nerve. (F1) Vestibular nuclei. (G1) Nucleus of the hypoglossal nerve. (H1) Nucleus alae cinerae. (I1) Restiform body.

With these anatomical data before us, let us now turn our attention to the nature of the expanding lesions which are likely to occur in that region. This will enable one to understand more clearly the variations in the effects of such lesions

upon the structures mentioned. It is quite reasonable to assume that lesions which are entirely extramedullary, that is to say, such as are almost entirely separated from the brain stem and affect the brain stem only secondarily, will act in a way somewhat different from expanding lesions which either arise from the brain stem in that region or invade that region secondarily from an extramedullary source of origin. Of course, not all angle tumors are extramedullary in the way the true "*acousticus*" tumors are. The latter are well encapsulated and thus technically can be enucleated without leaving permanent marks of disruption on the adjacent brain

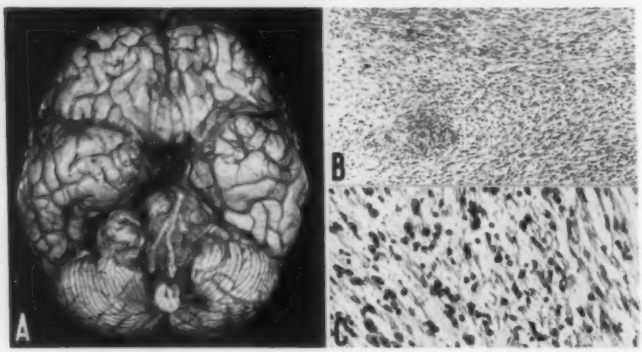

Fig. 3. (a) Base of the brain, showing typical acoustic neurinoma. (b) The stream-like arrangement of Schwann membrane cells. (c) Schwann membrane cells under higher magnification.

tissue. Such tumors, however, constitute only a quota of all tumor varieties occurring in the same location, for, in addition to the neuromas, there are neoplasms such as meningiomas, gliomas, sarcomas, metastatic carcinomas, and other expanding lesions such as aneurysms and granulomas.

The anatomical character and behavior of the true acoustic neuromas are to be considered first. These tumors are nodular (see Fig. 3, a), seemingly encapsulated, distinctly separated from the adjacent brain stem, except for a small area in the region of the entrance of the acoustic nerve. Histologically, they present a fairly uniform structure, not unlike that seen in neurofibroma of peripheral nerves (see Fig. 3, b, c), and include the palisading of nuclei, the presence of glial cells and streaming fibrous bands and occasionally of structures indicating the presence of embryonal (ependymal, vascular) rests.

In marked contrast to this type of tumor is the meningioma with its predominantly vascular components in one or another stage of differentiation, while the glioma is characterized by its infiltrating character. The histologic structure of sarcomas and other expanding lesions are too well known to require description.

Proceeding from this rather hasty survey of the histological features of tumors in the pontofacial angle to a discussion of their clinical features (see Chart 1), we come upon one of their early symptoms, but one which probably is not often correctly evaluated, that is, impairment of *hearing*. Attention may first be drawn to this defect by the appearance of *tinnitus*. It is this annoyance which often appears as the only symptom, inviting investigation and leading to the discovery of some impairment of hearing. Under such circumstances careful examination may bring to the fore the important observation that the patient is suffering from a "perception" disturbance; that is, from a *nerve deafness*. This, when recognized, should immediately arouse the suspicion of the possible existence of a tumor affecting the acoustic nerve. After a certain lapse of time and not infrequently after a few months or a longer period, the patient may become subject to recurrent attacks of *headache*. This symptom, while not always a dominant symptom in acoustic neurinoma, since this form of tumor grows rather slowly over long periods of time and does not, at first, cause any great disturbance in the circulation of the cerebrospinal fluid or provoke much increase in intracranial tension, the most probable cause of headache. But it, nevertheless, may pre-exist other symptoms and may continue throughout the clinical course, and hence must be regarded as highly significant.

In the course of time a new series of symptoms appear: an extremity may manifest a certain amount of awkwardness of a hand or arm on the side of the impaired hearing, or an awkwardness of the leg on that same side. This may be accompanied by apparent weakness or a feeling of weakness in the extremity affected which progresses to such an extent that the patient soon finds it difficult to use such extremity or extremities. Of course, the cause of these disturbances is quite obvious — it is due to an impairment in the function of either the cerebellar pathways coursing

through the medulla, a cerebellar peduncle or the cerebellum itself, caused by pressure of the neighboring expanding lesion. Of course, the vestibular nuclei situated in the angle between the cerebellum and the medulla may in part be responsible for such a disability.

There is already sufficient cause for the suspicion of the existence of an intracranial neoplasm and, no matter what its location, one is prompted to look for changes in *optic discs*. But here again, because of the slow growth of these tumors and because of the gradual adaptation of the brain to such a growth *papilloedema*, the most reliable sign of increased intracranial tension, is often not noted until the very end, and even then it may sometimes be lacking, in spite of the marked dislocation of the brain stem caused by the tumor. However, an early appearance of papilloedema is not unlikely and its conversion into a postneuritic atrophy at the height of the clinical picture is not an infrequent occurrence.

Other signs of increased intracranial tension, both of general and local origin, such as *nausea* and *vomiting* and *bradycardia* and such as *aphonia* and *dysphagia*, may also be lacking, unless the clinical picture reached its height and when they are provoked by direct pressure on some of the medullary centres and the vagus nerve. However, while the absence or mildness of these symptoms may be misleading, a thorough neurological examination will reveal sufficient evidence to identify the existence of a lesion affecting the acoustic nerve. Then a constellation of signs is discovered which indicates the involvement of the peripheral segment of the facial nerve; the vestibular and cochlear divisions of acoustic nerve; the disturbance in the cerebellar pathways and the posterior longitudinal bundle, causing nystagmus, unsteady gait and adiadokocinesis. When these findings are further supported by the results of the cochlear and vestibular tests, then there is, of course, little need for hesitation in arriving at the conclusion that an expanding lesion exists in the pontofacial angle and that it is probably in the nature of an acoustic neuroma. To recapitulate, the constellation of signs characterizing a tumor in the angle are: *1.* impairment of hearing or loss of hearing on the ipsilateral side, the deafness being of the perception or nerve type; *2.* peripheral facial paresis or

paralysis on the side of the suspected lesion; *3.* nystagmus indicating involvement of the cerebellar pathways or the posterior longitudinal bundle; *4.* equilibratory disturbances manifested in unsteady and broad-based gait, tendency to sway or fall to the affected side; clumsy use of the homolateral upper extremity and a moderate atonia or hypotonia on the side affected, due to the involvement of the cerebellar pathways; *5.* moderate pyramidal tract signs and sensory changes on the side contralateral to the site of the tumor whenever compression of the brain stem becomes pronounced; *6.* sensory disturbance in the distribution of the trigeminal nerve on the homolateral side, with subjective pain associated with hypalgesia, hypesthesia and corneal anesthesia on the affected side. Frequently there is also noted a VIth nerve paresis, but the abducens, because of its very long course, is a very vulnerable structure and is affected by any lesion of the posterior fossa. Since it is even found implicated in supratentorial lesions, it is often of doubtful focalizing significance. With the involvement of the VIth nerve, diplopia is quite likely to occur. Unequal pupils are frequently noted and should not lead one astray from the correct diagnosis.

This picture holds true for the fairly typical instance of pontofacial angle tumor in which the tumor is derived from the sheath of the acoustic nerve and does not invade the adjacent brain stem but brings about a great deal of distortion of its external configurations and some displacement of internal structures. It is highly significant to note that in spite of this marked distortion of external form and internal structure of the brain stem, there is commonly very little disintegration within the interior of the brain stem. As a matter of fact, in four cases of acoustic neuroma studied by me, in which the stem has been cut serially and studied by appropriate methods, no disintegration of internal structures, such as demyelinization of tracts or nuclear destruction, could not be detected. This would indicate that the pressure exerted by an external compressing agent such as an acoustic tumor, causing a disturbance in the function of the internal structures (nuclei and tracts), does it without causing permanent damage to the tissue. Hence, with the removal of the expanding lesion these structures may assume a normal position and function is thus restored. This is readily supported by clinical evidence that when patients with acoustic

tumors are relieved of the tumor, many of the functions, with the exception of hearing, are restored.

These circumstances differ for extramedullary tumors with invasive tendencies or for tumors which arise within the brain stem, as will be apparent from a discussion of the features which characterize tumors occurring in this regior., but such which invade or take origin in the brain stem adjacent to the angle. They are the pontofacial angle tumors other than acoustic neuromas. Among them are the meningiomas, tumors which approach closest the symptomatology of true acoustic tumors, as exemplified by the following case. Here, it should be noted that the anatomical differences are more of an academic interest since this extramedullary tumor in its behavior and its response to surgery is very similar to acoustic neuroma:

Case 1: Tinnitus in the left ear of two years' duration; pain in the left cheek of one year duration followed by the development of unsteady gait, papilloedema, nystagmus, left peripheral facial paralysis, impaired hearing on the left side and other signs of brain stem involvement. Tumor of left middle fossa diagnosis. Necropsy: Pachymeningioma of left pontofacial angle.

History: (Adm. 315202. P.M. 7249). The patient, a woman, age 52 years, was for a period of two years subject to a buzzing sound in her left ear. One year later she began to experience pain in her left cheek. The pain gradually spread to the jaw, eye and forehead on the same side. Soon thereafter, her gait became unsteady and she could not walk without support. She did not complain of headache or visual disturbances and did not vomit.

Examination: There was but slight blurring of the margins of the discs. Spontaneous nystagmus was present on the left lateral gaze. The left upper eyelid was drooping slightly. There was analgesia, anesthesia and thermoanesthesia in the distribution of all the three branches of the left trigeminal nerve. The lower jaw was deviated to the left. Paresis of the left external rectus and a left peripheral facial paralysis was also present. Hearing was impaired on the left side. Power in all extremities was objectively intact. There was generalized hyper-reflexia, with the deep reflexes being more active

on the right side. A Babinski sign was elicited on the right side. All of the abdominal reflexes were absent. The gait was guarded, with a tendency to fall to the right. A coarse, irregular asymmetrical tremor was present in all extremities.

Course: A diagnosis of a tumor in the left middle fossa was made, but while under observation the patient developed pneumonia and died soon thereafter.

Necropsy Findings: A tumor about the size of a plum, white, firm, sharply circumscribed from the brain substance, though somewhat adherent, was found in the left pontofacial

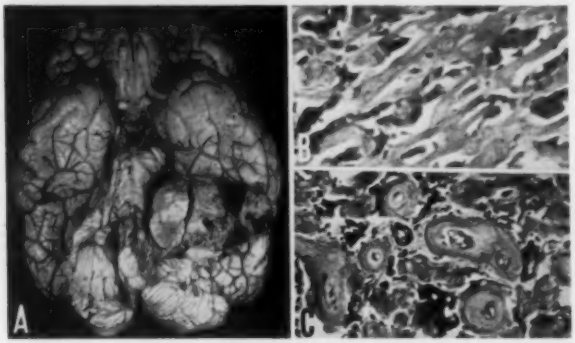

Fig. 4. (a) Base of brain, showing a meningioma in the pontofacial angle (Case 1). (b) Collagenous fibres indicating the pachymeningiomatous nature of the tumor (Case 1). (c) Hyalinized blood vessels in the depth of the tumor.

angle (see Fig. 4, a). It compressed the left side of the pons and the adjoining medulla. On its lateral aspect, the tumor was intimately connected with the overlying dura.

Microscopic: Examination of the tumor disclosed it to be in the nature of a pachymeningioma (see Fig. 4, b, c).

Comment: In this case a meningioma, of the benign variety, situated in the pontofacial angle simulated clinically an acoustic aneuroma. The involvement of the trigeminal nerve, with both the motor and sensory portions being affected, though somewhat atypical, is, nevertheless, not uncommonly found in true acoustic neuromas. Here, of course, it is obvious that the histological character of the tumor does not militate against surgical intervention.

It should not be lost sight of that meningiomas are of several varieties, the least troublesome being the so-called pachymeningioma, as in Case 1, or leptomeningiomas, while others, such as the pial or hemangiomatous meningioma, may approach the invasive type of tumor, as exemplified by Case 2. In this case the mode of onset of symptoms and the more stormy and relatively shorter clinical course are conditions obviously due to the greater vascularity of this type of tumor.

Case 2: Sudden onset of suboccipital pain, vomiting, vertigo, bilateral papilloedema, spontaneous nystagmus, left facial paresis, left cerebellar signs and other manifestations of brain stem involvement. Unsuccessful exploration. Necropsy: Hemangioendothelioma of left pontofacial angle.

History: (Adm. 312564. P.M. 7205). The patient, a young man, age 22 years, passed through a brief but rather stormy illness of six weeks' duration, at the age of 19 years. He then complained of pain in the back of the neck, double vision, photophobia and weakness in the lower extremities. His illness was diagnosed as lethargic encephalitis. He made an apparently complete recovery and for two and one-half years remained apparently well, when one morning he awoke with severe pain in the suboccipital region, which was intensified by movement of the head; he vomited several times and on walking he would become dizzy and would stagger to the right. The attacks of vomiting and vertigo occurred repeatedly during the first three weeks and became more frequent during the following three weeks. He entered the hospital on March 24, 1930.

Examination: There was slight bilateral early optic neuritis, more marked on the left side; slight inequality of pupils, with the right being larger than the left; normal pupillary reactions; some weakness of the left external rectus; spontaneous nystagmus to the left; diplopia with eyes in left lateral fixation; and slight facial weakness on the left side. The left palate and posterior wall of the pharynx were analgesic. Power in all extremities was intact. There was some resistance to forward flexion of the head. The right triceps reflex was somewhat more active than the left. The left knee jerk was more active than the right. Moderate ataxia was noted in the left upper and lower extremities, and

adiadokocinesis was elicited on the left side. His gait was poorly co-ordinated and he would fall on rapid turning to the left.

Course: A diagnosis of a posterior fossa lesion involving the left cerebellar hemisphere and adjacent structures was made. The lesion was thought to be expanding in character and most likely a hemangioma. A degenerative lesion in the nature of multiple sclerosis was also postulated if the disc findings after further observation, should show no further progression. In the course of time, the tongue and jaw were found to deviate to the right, and bilateral corneal hyperesthesia developed. Somewhat later, paralysis of the left recurrent laryngeal appeared; the tongue now deviated to the left. It was then thought that the lesion was intramedullary.

A craniotomy was attempted, but the patient did not stand the operation well. Gradual decline with the development of new signs; atrophy of the left side of the tongue, more marked diplopia, advancing papilloedema with retinal hemorrhages.

A second craniotomy was tried but could not be completed because of the poor condition of the patient, and a third attempt failed similarly. The patient declined rapidly and died.

Necropsy Findings: On exposing the base of the brain, a tumor mass was encountered in the region of the left cerebellopontine angle. The tumor appeared to be both extramedullary and intramedullary in location and occupied the mesial portion of the inferior surface of the left cerebellar lobe, causing a very marked distortion of the brain stem, displacing it to the right. The tumor mass extended down into the foramen magnum and the upper part of the cervical canal, thus causing a marked compression of the spinal cord in that region. It had a very shiny surface, was firm and could be partly dissected away from the brain substance, being at some points intimately connected with it. The cranial nerves from the IXth to XIIth, inclusive, on the left side could not be identified as they appeared to be embedded in the tumor substance.

On sectioning the brain, the tumor was found to be about 4 cm. in diameter. It projected from the left cerebellar hem-

isphere on to the brain stem, compressing it and displacing it to the right (see Fig. 5). There was no direct invasion of the brain stem itself. The tumor mass was solid in parts and in areas cystic, filled with gray gelatinous material.

Comment: In this case, the clinical features would justify exploration, with the view that a benign pontofacial angle tumor might be encountered. It is true that this meningioma, being a little different in type than the previous case, offers a more difficult surgical problem. However, with the recognition of such a difficulty, craniotomy may be undertaken with some promise of a satisfactory result.

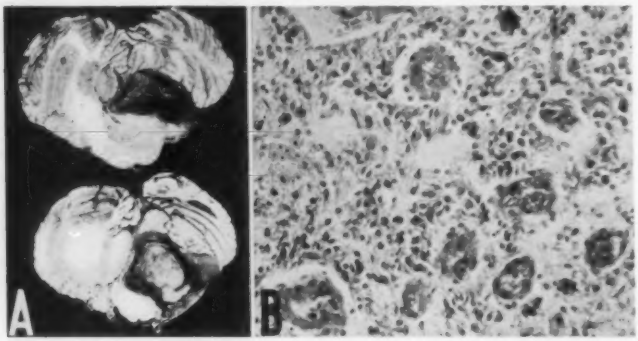

Fig. 5. (a) Cross-section of the brain stem, showing the invaginated tumor in the pontofacial angle (Case 2). (b) The histological character of the tumor (Case 2).

Similarly, in another type of pontofacial angle tumor, this time not neoplastic in character, though also expanding in character, but such as an aneurysm of the vertebral artery, the clinical picture deviates more strikingly from that of the classic syndrome of acoustic neuroma. The clinical behavior of this lesion, being fully determined by its anatomical character, is probably explained by the fact that the changes in the involved arterial trunk provoke alteration in the field in the brain stem fed by its branches.

Case 3: Sudden onset of severe pain in the left side of the neck, recurrent headache, dysphagia, dysphonia, vertigo, bilateral papilloedema, horizontal nystagmus, deviation of the jaw to the right, hypertension. Suboccipital exploration. Necropsy: Aneurysm of the left vertebral artery.

History: (Adm. 421780. P.M. 10754). The patient, a woman, was admitted to the hospital for the first time in 1926, at the age of 36 years, for a postpartem sepsis and bronchopneumonia. She made a good recovery and passed through a normal pregnancy two years later. (The child, however, died at the age of 10 days, presumably of pneumonia.) One year later, she had a miscarriage and since then no pregnancies. At the age of 42 years (in 1932) she developed severe drawing pains in the left neck, which did not radiate and were not related to movement of the head. With that, she became very "nervous." At about the same time she had a slight dizzy spell, during which, for a few seconds, everything went black before her eyes. She was found to have hypertension, but, nevertheless, her symptoms were thought to be sinusitis, and treatment for this condition resulted in slight relief from the pain in the neck. She soon, however, developed recurrent headache, with its maximum point of intensity over the right supraorbital region. These headaches lasted for hours at a time and were relieved only by medication. Shortly before her admission to the hospital she developed difficulty in swallowing, a change in the timbre of her voice and intelligibility of her speech. Generalized weakness set in and dizziness and sleepiness occurred in attacks. She would regurgitate food and had a feeling of fullness in her chest. "Drawing pains" in both calves made it difficult for her to walk or support herself (the patient had had varicose veins for many years and was treated with injections and wore elastic stockings without relief). Frequency of urination and nocturia had also been present. She was admitted to the hospital on March 23, 1938.

Examination: Sense of smell was diminished bilaterally. There was moderate bilateral exophthalmos. Bilateral papilloedema and postneuritic optic atrophy were present. There was horizontal nystagmus in both lateral planes. The left palpebral fissure was wider than the right. The corneal reflexes were bilaterally inactive; the jaw was deviated to the left; her voice was hoarse; the palate lagged on innervation and the uvula deviated to the left. There was some weakness of the left arm and leg, with a bilateral tendency to pronation and convergence of the outstretched arms. The tendon reflexes were generally hyperactive, but equal. The superficial reflexes were depressed. Vibratory sense was

diminishd in the lower extremities. The gait was broad-based and both legs dragged, the left more than the right. The blood pressure was 165 systolic and 120 diastolic.

Laboratory Data: The cerebrospinal fluid was clear, colorless, containing three cells and under an initial pressure of 108 mm. of water. The Ayala index was 5.5. The Pandy test was 4+. The total protein was 64 mm. per cent. X-ray examination of the skull revealed no evidence of increased intracranial pressure, and a calcified pineal body was in normal position. X-ray examination of the sinuses showed no evidence of disease. A ventriculogram revealed marked bilateral dilatation of the lateral ventricles, the right being slightly larger than the left. There was no deviation from the midline and the third ventricle appeared dilated; the iter was visualized. Caloric tests were carried out and were reported as follows: "The findings are suggestive of a posterior fossa lesion. The inward pastpointing of the left hand and the failure to pastpoint to the left on stimulating the left vertical canals suggest a lesion with its greater part in the left posterior fossa." A laryngoscopic examination showed a definite palatal paralysis.

Course: An impatentorial midline lesion was diagnosed, and a suboccipital exploration was attempted. It had to be discontinued, for the patient's condition became alarming. She remained in stupor for several days and after a brief and slight recovery declined again and died four days after the attempted craniotomy.

Necropsy Findings: In the course of the removal of the brain and during the attempt to separate the medulla from its attachments to the base of the skull, a large bluish mass was revealed on the left side. It appeared to compress the inferior surface of the cerebellum and the left half of the medulla and pons, and extended into the foramen magnum. Upon removal this mass, measuring about $1\frac{1}{2}''$x1''x$\frac{3}{4}''$, was easily shelled out from the dell it had formed and found to be a large aneurysm of the left vertebral artery (see Fig. 6). It pressed into the pons, pushing it upward and to the right, and displaced the medulla and upper part of the cervical cord towards the right, with a resultant marked deformity. The accessory nerve was stretched over the mass.

On sectioning, the ventricular system was found markedly dilated as far as the posterior end of the aqueduct of Sylvius. At that point, the aqueduct was displaced to the right and reduced to an oblique narrow slit. The bed of the aneurysm consisted of the left cerebellar hemisphere, with the pons above it and the medulla oblongata to the right. All of these three structures were compressed and somewhat displaced, but the medulla was the one which had suffered most. From the border of the pons down to the mergence of the medulla with the spinal cord, it was flattened and displaced to the right.

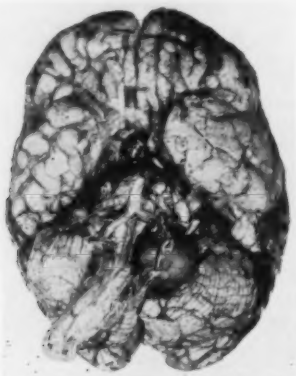

Fig. 6. Base of the brain, showing aneurysm of the vertebral artery, located in the pontofacial angle (Case 3).

Comment: The attempt of suboccipital exploration was, I believe, justified here, even though it may have contributed to the earlier fatal issue. Were the patient's condition such as to allow exposure and recognition of the lesion, the removal of the aneurysm is not out of reach of the competent neurosurgeon.

Another, but less common, lesion in the angle is the metastatic carcinoma. This is an example of an expanding lesion whose behavior, in spite of its location, is decidedly different from that of an acoustic neuroma. Here, the precipitant onset, the freedom from cochlear and vestibular symptoms, the intense headache and distressing pain in the neck are important diagnostic leads.

Case 4: Cerebral manifestations of four weeks' duration; acute onset, intense headache, vertiginous attacks, Bruns' sign, other signs of increased intracranial tension without papilloedema, dissemination of objective neurolgic signs, sudden death without operative intervention. Necropsy: Metastatic carcinoma in the left pontofacial angle.

History: (Adm. 258521. P.M. 5154.) J. Y., a man, age 39 years, married, was admitted to the hospital complaining of rapid loss of weight for the preceding four weeks, of intense headache and general weakness for three weeks, with attacks of vomiting for one week. He was losing weight rapidly and his strength was declining. He complained of constant headache, with sharp shooting pain in the occiput. The headache and pain were frequently precipitated by sudden movements of the head. As the headaches had become more intense they were followed by frequent attacks of vomiting, associated with vertiginous episodes. During such episodes he was disturbed by perceiving objects running before him from right to left; in response to them he would incline his body to the left. More recently the pain in the head became more intense and was associated with fainting spells lasting for several minutes.

Examination: The patient was acutely ill and apparently in pain. He showed unequal pupils, the left larger than the right; increased knee jerk and a suggestive Babinski sign on the left. No abnormal masses were palpable and no points of tenderness were noted.

Course: On the day after admission, slight nystamus on left lateral gaze and bilateral ptosis were noted; also a tendency toward a bilateral Kernig sign without rigidity of the neck. A lumbar puncture yielded clear fluid under normal pressure with 15 cells per cubic millimetre. The temperature ranged from 98° to 99° F. No abnormal changes in the fundi were observed at any time. In the absence of papilloedema, meningoencephalitis was regarded as the most probable diagnosis; however, it was thought that a cerebral neoplasm could not be ruled out entirely. The patient died suddenly without warning, eight days after admission.

Necropsy Findings: Gross Anatomy: The dura was normal. The pia-arachnoid was normal except for a small area

on the dorsal surface of the left cerebellar lobe, where it was unusually smooth and glistening but thick and adherent to the underlying cortex. The brain was of average size and of normal consistency, except for the area on the dorsal surface of the left cerebellar lobe, underlying the thickened pia-arachnoid, where it was soft and fluctuating. On the under surface of the cerebellar hemisphere at the left pontofacial angle there was a small nodule, 1 cm. in diameter, which was

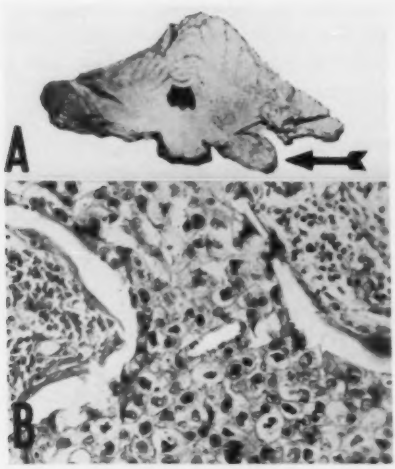

Fig. 7. (a) Section of brain stem, showing a metastatic nodule in the pontofacial angle (Case 4). (b) The carcinomatous structure of the tumor.

hard and easily shelled out (see Fig. 7, a). On section it had a granular appearance. A transverse midcerebellar section showed a similar, but somewhat larger mass, 2 cm. in diameter. It was also granular but much softer in make-up, particularly in its centre.

Microscopic Anatomy: Section through the tumor mass in the cerebellum showed that the predominating type of cell was of the large squamous type, with considerable cytoplasm and large central nucleus. The cells were arranged in sheets surrounding large blood vessels. Numerous mitotic figures were seen, indicating rapid growth. Large areas of necrosis alternated with zones of rapidly proliferating cells. At the

periphery of the tumor, the blood vessels showed dense perivascular infiltrations with small round cells, a condition often seen in neoplastic lesions of the brain, indicating a tissue reaction to the foreign (tumor) invader (see Fig. 7, b). The final diagnosis was that of a metastatic squamous-cell carcinoma.

Comment: In this case, the diagnosis of meningoencephalitis was in the foreground. The acute onset, the absence of decided localizing signs and the slight rise in temperature and the presence of 15 cells in the cerebrospinal fluid justified the assumption of an inflammatory lesion in preference to one of a neoplastic character; however, the headache associated with frequent attacks of nausea and vomiting, the scarcity of localizing signs and the rapidity with which the clinical picture unfolded itself could suggest the neoplastic character of the lesion and its metastatic nature.

It is striking that the cerebellum was singled out as the only seat for the metastatic tumor. This localization, however, fully explains the clinical picture, for it caused bilateral, symmetrical internal hydrocephalus. The latter, as is well known, may cause symptoms which can simulate a diffuse inflammatory lesion of the brain, as well as give rise to signs of meningeal irritation. The location of the tumor also will explain the Bruns' syndrome in the case. It is, however, difficult to account for the absence of changes in the discs.

Among the tumors which are most likely to take root in the brain stem adjacent to the pontofacial angle are the gliomatous neoplasms, of which the following is a good example:

Case 5: Headache, vertigo, ataxia, bilateral papilloedema, left facial paresis, left adiadokocinesis, nystagmus. Postoccipital exploration. Necropsy: Spongioblastoma, left pontofacial angle.

History: (Adm. 255330. P.M. 5072). The patient, a young man, age 23 years, became subject to frontal headaches occurring at intervals of two to three weeks. At the end of one year, they became more frequent and were accompanied by dizziness. In another few weeks, his gait became ataxic, he was forced to hold his head back in walking. He had occasional nausea, but no vomiting. He entered the hospital on May 21, 1925, one year and one-half after the onset of symptoms.

Examination: There was a bilateral papilloedema (see Fig. 4, d); bilateral corneal anesthesia; weakness of the lower half of the left face; slight deviation of the tongue to the left. His gait was unsteady on a broad base with swaying to either side, with some tendency to fall backwards. He carried his head tilted to the left, with the chin turned to the right. There was slight bilateral ataxia in finger to nose tests and a questionable left adiadokocinesis. He pastpointed slightly with both hands, but more so with the left. The deep reflexes were hyperactive. The knee jerks were somewhat pendulous. The left palpebral fissure was possibly greater

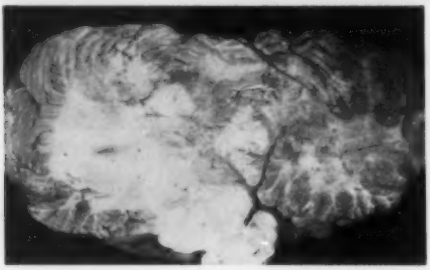

Fig. 8. Section of the brain stem, showing the invasive character of the gliogenous tumor in the pontofacial angle (Case 5).

than the right. There was horizontal nystagmus on lateral gaze, more to the left, and slight rotary nystagmus on looking upward.

Laboratory Data: The cerebrospinal fluid, blood and urine tests were all negative. X-ray examination of the skull showed atrophy of the sella and erosion of the clinoids. Otological examination disclosed absence of pastpointing when stimulating the left horizontal and vertical canals (nystagmus normal), indicating an interference with the fibres of the vestibulocerebello-pathway.

Course: The lesion was thought to be probably in the left cerebellar hemisphere, but the vertical nystagmus pointed also to an involvement from pressure upon the brain stem. An attempt was made to do a posterior occipital craniotomy. Bleeding was profuse and the patient's condition did not allow complete operation. Following the operation the patient devel-

oped signs of medullary compression. These cleared up but signs of pneumonia set in and the patient died in hyperpyrexia.

Necropsy Findings: Gross inspection of the brain shows marked flattening of gyri, marked hypertrophy and induration of left cerebellar lobe, vermis of the left middle cerebellar peduncle, due to an infiltrative neoplasm. The medulla at its upper part is edematous, swollen and softened (see Fig. 8).

Sections of the tumor showed typical histological picture of spongioblastoma multiforme. Numerous flask-shaped cells, multinuclear giant-like rosette formations characterized the tumor.

Comment: Here, in spite of the fact that the focalizing signs pointed to a left pontofacial angle tumor, the absence of impairment of hearing spoke against an acoustic neuroma; moreover the caloric tests indicated a stem involvement. The marked elevation of discs to the right also have been considered as suggesting a lesion infiltrating the brain stem and hence surgical intervention under such circumstances should be weighed very carefully.

SUMMARY.

I have come to the end of my demonstration and in summarizing offer the following: Acoustic neuroma bear clinical earmarks which when carefully looked for can be readily recognized. Thus, a correct diagnosis need never be missed and the patient can be most often assured of successful surgical intervention.

Tumors other than acoustic neuroma are quite numerous. Among them are the leptomeningiomas, which behave very much like acoustic neuromas; there are also the hemangiomas, gliomas, sarcomas and others whose behavior is quite often so different that it may permit the recognition of their character and thus often reveal contra-indications for surgical interference. The most important contribution to the solution of the problem in the differential diagnosis can be made by the otologist employing the now available excellent methods of investigating both the cochlear and vestibular mechanisms.

1133 Fifth Avenue.

IN MEMORIAM

JOHN B. RAE, M.D.,

1867-1941.

Dr. John B. Rae, formerly of New York City, died at the home of his sister in Douglas, Ariz., Nov. 20, at the age of 74 years.

In 1895, Dr. Rae received his M.D. degree from Glasgow University and immediately began the practice of his specialty. He was at one time Professor of Otolaryngology at the New York Post-Graduate Hospital, and at the time of his death was vice-president of the Manhattan Eye and Ear Infirmary, and consulting aural surgeon at the Bronx Eye and Ear Infirmary.

He served with the Post-Graduate Hospital unit at Base Hospital No. 8 during the World War with the rank of Captain.

In 1924, Dr. Rae was honored with the presidency of the American Otological Society. He was a Fellow of the American Laryngological, Rhinological and Otological Society and of the American College of Surgeons, and during his lifetime made many valuable contributions to the literature of his special field of medicine.

DR. JEAN J. E. MOURE,

1855-1941.

Dr. Jean J. E. Moure, the foremost French laryngologist, died Dec. 2, 1941, at Cannes on the Riviera, at the age of 86 years.

Dr. Moure was internationally recognized and honored for his outstanding achievements in otorhinolaryngology and he made many valuable contributions to its literature.

For many years Dr. Moure was Clinical Professor of Otorhinolaryngology at Bordeaux University. Under his directorship this department became famous over a period of 40 years and students from many nations received the benefit of his teachings there.

In 1880, he founded and edited the *Revue de Laryngology, Otology and Rhinology*, relinquishing these duties only a few years ago to his son-in-law, Dr. Georges Portmann.

The presidency of the International Congress on Otology was bestowed upon Dr. Moure in 1904; he was a Commander of the French Legion of Honor, a Chevalier of Christ of Portugal; from the former King Alfonso XIII he received the Grand Cross of Isabella the Catholic, and membership in the Order of Alfonso XII.

Through the efforts of this eminent otolaryngologist, laryngology was transferred from the medical to the surgical field and he was famous for his operations. The younger generation of otolaryngologists will find their tasks much easier due to the unselfish and untiring efforts of Dr. Moure.

BOOK REVIEW.

Voice and Articulation Drillbook. By Grant Fairbanks. Harper and Brothers, 1941. Cloth, 234 pp.

This volume, an outgrowth of extensive experimental efforts at the University of Iowa, is intended as a basic source of drill material for individual clinical work in speech correction and improvement. In intelligent hands it lends itself to use in self-help and self-analysis, although in parts it is somewhat too professional in tone for the lay individual. Fundamentals of phonetics and articulation are clearly presented, accompanied by drills for ready application. Examination forms for objective evaluation and analysis of voice and articulation are an attractive feature. In the consideration of voice disorders a nice distinction is made between those factors concerned with the original tone as initiated by the vocal cords and those factors concerned with the selected modification of this tone by the resonance cavities. The emphasis in the discussion of voice quality is placed on the former set of factors. The author might have enhanced the value of the volume to the practitioner if he had devoted more attention to that elusive characteristic of speech-voice quality. This shortcoming has characterized many previous works on speech correction and speech improvement. The speech clinician, however, can find numerous helpful suggestions of a practical nature in Dr. Fairbanks' book.

S. R. S.

DIRECTORY OF NATIONAL OTOLARYNGOLOGIC SOCIETIES.

American Otological Society.

President: Dr. E. M. Seydell, 107 W. Douglas Avenue, Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73rd Street, New York.
Place: Atlantic City, May 28-29, 1942.

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American Broncho-Esophagological Association.

President : Dr. W. Likely Simpson, 899 Madison Avenue, Memphis.
Secretary: Dr. Paul Holinger, 1150 N. State Street, Chicago.
Place: Atlantic City, June 8-9, 1942.

American Laryngological Association.

President: Dr. T. E. Carmody, 227 16th Street, Denver.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th Street, New York.
Place: Atlantic City, May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbitt, 1912 Spruce Street, Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Avenue, New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester, N. Y.
Place: Atlantic City, June 1-3, 1942.

Sections:

Eastern—Chairman: Dr. Henry P. Johnson, 32 Deering Street, Portland, Maine. Jan. 15, 1942.
Southern—Chairman: Dr. William L. McDougall, 915 Doctors' Building, Atlanta. Jan. 23, 1942.
Middle—Chairman: Dr. William E. Sauer, 3720 Washington Boulevard, St. Louis. Jan. 21, 1942.
Western—Chairman: Dr. Emil F. Tholen, 113¢ W. 6th Street, Los Angeles. Jan. 31-Feb. 1, 1942.

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology.

Chairman: Dr. Gordon F. Harkness, 215 Main Street, Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust Street, Philadelphia.

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CONTENTS

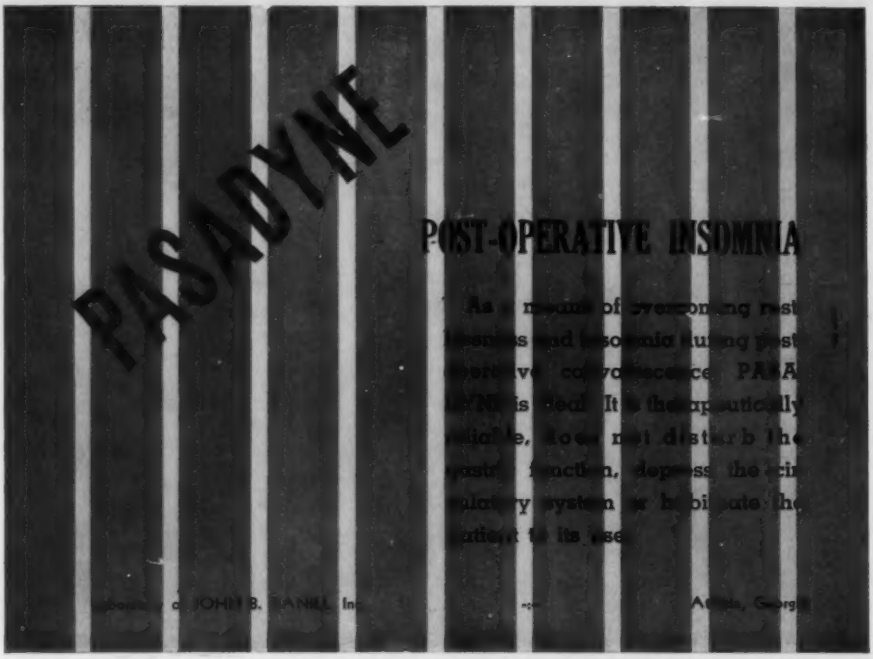
Books Received

Books and monographs received are acknowledged in this column. This notice may be regarded as a return courtesy to the publisher or author. Reviews will be published later as the editors may elect.

Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

DISEASES OF THE NOSE, THROAT AND EAR. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E. Surgeon to the Royal Infirmary, Edinburgh; Lecturer in Diseases of the Nose, Throat and Ear, University of Edinburgh. Second Edition with 446 pages, including index, 74 illustrations in black and white and four colored plates. Edinburgh: E. and S. Livingston, 16 and 17 Teviot Place. 1941. Price 15 s net; postage 7 d.

SINUS. By Russell Clark Grove, M.D., Chief of Ear, Nose and Throat Department of the Allergy Clinic, Roosevelt Hospital, New York. One hundred eighty-four pages, including index and eight illustrations. New York: Alfred A. Knopf, Inc. 1941. Price $2.00.



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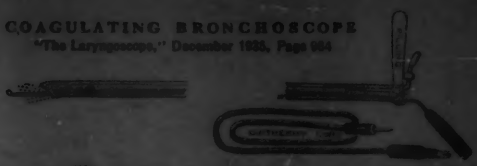
"The Laryngoscope," December 1935, Page 984



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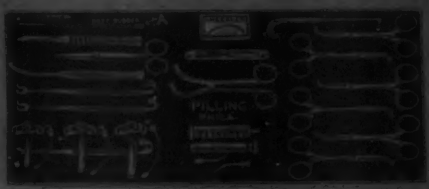

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